Indices and Implications of Emotional Underarousal for Persons with a History of Head Trauma

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Abstract

We examined the role of altered emotional functioning across the spectrum of injury severity (mild head injury [MHI], moderate/severe traumatic brain injury [TBI]), its implications for social behaviours, and the effect of modifying arousal and its relation to cognitive performance. In the first study (N = 230), students with self-reported MHI endorsed engaging in socially unacceptable and erratic behaviours significantly more often than did those with no MHI. We did not find significant differences between the groups in the measure of emotional intelligence (EI); however, for students who reported a MHI, scores on the EI measure significantly predicted reports of socially unacceptable behaviours such that lower scores predicted poorer social functioning, accounting for approximately 20% of the variance. Also, the experience of postconcussive symptoms was found to be significantly greater for students with MHI relative to their peers.

In the second study (N = 85), we further examined emotional underarousal in terms of physiological (i.e., electrodermal activation [EDA]) and self-reported responsivity to emotionally-evocative picture stimuli. Although the valence ratings of the stimuli did not differ between students with and without MHI as we had expected, we found evidence of reduced and/or indiscriminate emotional responding to the stimuli for those with MHI which mimics that observed in other studies for persons with moderate/severe TBI. We also found that emotional underarousal followed a gradient of injury severity despite reporting a pattern of experiencing more life stressors.

In the third study (N = 81), we replicated our findings of emotional underarousal for those with head trauma and also uniquely explored neuroendocrine aspects (salivary cortisol; cortisol awakening response [CAR]) and autonomic indices (EDA) of emotional

dysregulation in terms of stress responsivity across the spectrum of injury severity (MHI [*n* = 32], moderate/severe TBI [*n* = 9], and age and education matched controls [*n* = 40]). Although the manipulation was effective in modifying arousal state in terms of autonomic and self-reported indices, we did not support our hypothesis that increased arousal would be related to improved performance on cognitive measures for those with prior injury. To our knowledge, this is the only study to examine the CAR with this population. Repeated measure analysis revealed that, upon awakening, students with no reported head trauma illustrated the typical CAR increase 45 minutes after waking, whereas, students who had a history of either mild head trauma or moderate/severe TBI demonstrated a blunted CAR. Thus, across the three studies we have provided evidence of emotional underarousal, its potential implications for social interactions, and also have identified potentially useful indices of dysregulated stress responsivity regardless of injury severity.

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List of Abbreviations

ABI	Acquired Brain Injury
ACRM	American Congress of Rehabilitation Medicine
ACTH	Adrenal Corticotrophin Hormone
ANCOVA	Analysis of Covariance
ANOVA	Analysis of Variance
ASC	Altered State of Consciousness
CAR	Cortisol awakening response
CDC	Center for Disease Control
CIHI	Canadian Institute of Health Information
CIHR	Canadian Institute of Health Research
CRH	Corticotrophin Releasing Hormone
CT	Computerized Tomography
DAI	Diffuse Axonal Injury
DKI	Diffusional Kurtosis Imaging
DLPFC	Dorsolateral Prefrontal Cortex
DTI	Diffusional Tensor Imaging
EDA	Electrodermal Activity
EMG	Electromyography
fMRI	Functional Magnetic Resonance Imaging
GCS	Glascow Coma Scale
GR	Glucocorticoid Receptor
HPA	Hypothalamic-Pituitary-Adrenal
ICD	International Classification of Diseases
LOC	Loss of Consciousness
MHI	Mild Head Injury
mPFC	Medial Prefrontal Cortex
MR	Mineralocorticoid Receptors
MRI	Magnetic Resonance Imaging
MTBI	Mild Traumatic Brain Injury

MVC	Motor Vehicle Collision
No MHI	No Mild Head Injury
OFC	Orbitofrontal Cortex
OMPFC	Orbitomedial Prefrontal Cortex
PCD	Post-concussional Disorder
PCS	Post-concussion Syndrome
PET	Positron Emission Tomography
PFC	Prefrontal Cortex
PNS	Peripheral Nervous System
PTA	Post Traumatic Amnesia
PTSD	Post-traumatic Stress Disorder
PVN	Periventricular Nucleus
SNS	Sympathetic Nervous System
TBI	Traumatic Brain Injury
TSST	Trier Social Stress Test
VMPFC	Ventromedial Prefrontal Cortex
WHO	World Health Organization

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GENERAL INTRODUCTION

Overview

The complex cognitive, emotional and social sequelae following head trauma can be transient or prolonged (e.g., Lezak, Howieson, Bigler, & Tranel, 2012; Ponsford, 2013; Zasler, Katz, & Zafonte, 2007). It is generally acknowledged that acute and long-term impairments in cognitive and emotional functioning postinjury are commonly observed following moderate and severe traumatic brain injury (TBI; e.g., Iverson & Lange, 2009; Lezak et al., 2012; Stuss & Levine, 2002). The neurobehavioural sequelae (see McAllister, 2011) often pose barriers to social reintegration postinjury (e.g., Larsson, Bjorkdahl, Esbjornsson, & Sunnerhagen, 2013; Ponsford & Schonberger, 2010; Sloan & Ponsford, 2013). These cognitive and behavioural changes following neural disruption are often associated with impaired psychosocial functioning (e.g., Dawson, Levine, Schwartz, & Stuss, 2004; Draper, Ponsford, & Schönberger, 2007; Kringelbach & Rolls, 2004; Naqvi, Shiv, & Bechara, 2004; Stuss & Levine, 2002). Of particular interest to the current research is the role of altered emotional functioning (e.g., see Bechara et al., 2000; Hopkins, Dywan, & Segalowitz, 2002; Rogers & Read, 2007; Stuss & Levine, 2002) and its implications for socioemotional functioning (e.g., Leopold et al., 2011).

Emotional functioning is often disrupted after moderate or severe TBI, especially with frontal lobe trauma; several studies have demonstrated deficits in ability to recognize emotions (e.g., Bornhofen & McDonald, 2008; Ietswaart, Milders, Crawford, Currie, & Scott, 2008), altered socioemotional functioning (e.g., Milders, Fuchs, & Crawford, 2003; Ponsford & Schoenberger, 2010) and emotional responses after head trauma (e.g., Hopkins et al., 2002; Hornak, Rolls, & Wade, 1996; McLelland &

McKinlay, 2013). Altered emotional arousal has also been documented, which may be captured by the terms of *hyperarousal* (e.g., increased prevalence of anxiety disorders; increased emotional reactivity – e.g., Whelan-Goodinson, Ponsford, Johnston & Grant, 2009) and of *hypoarousal* (e.g., apathy; attenuated emotional responding/flattened affect – see McLelland & McKinlay, 2013; increased prevalence of depressive disorders – see Rogers & Read, 2007 for review of psychiatric comorbidity; reduced autonomic responses [i.e., electrodermal activation] to emotional stimuli – see Hopkins et al., 2002; Tranel, 2000).

Although much research has demonstrated that persons with moderate or severe TBI are often emotionally labile (e.g., overly reactive) and/or demonstrate an attenuated capacity for emotional experiences (e.g., Croker & McDonald, 2005; Hornak et al., 1996; McLelland & McKinlay, 2013), emotional functioning after milder head trauma has not been explored to the same extent. Questions remain regarding emotional functioning of persons with mild head trauma as well as whether emotional and psychosocial difficulties involve a continuum of severity of injury (i.e., more severe injury, more emotional functioning difficulties). This dissertation focused on examinations of the emotional, cognitive, and social sequelae after head trauma compared to persons with no reported prior head trauma with a particular focus on emotional arousal and its potential implications for overall functioning. The models of emotional volatility/lability or attenuated emotional responsiveness following TBI were examined in terms of emotional arousal (self-report and physiological) and overall emotional functioning. Physiological indices of emotional arousal (e.g., salivary cortisol, electrodermal activation, heart rate), self-reported experience of emotional events, and dysregulated stress responsivity (i.e.,

the cortisol awakening response [CAR]) were of primary interest especially as a function of injury severity (e.g., mild, moderate/severe TBI) to demonstrate a continuum of neural disruption (e.g., Alexander, 1995; Iverson & Lange, 2009) and, in some instances, a gradient of emotional responsivity. The following discussion highlights the literature that has guided this trajectory of research.

Structure of the Chapter

This chapter of the dissertation focuses on TBI in general. TBI is defined in the current state of knowledge, classifications of and grading of injury severity are presented, after which the epidemiology of TBI in North America will be discussed. The complex mechanisms and pathophysiology affecting the severity of head trauma is described, and, lastly, the presentation and assessment of neurobehavioural sequelae is discussed to provide a context for the dissertation research.

Traumatic Brain Injury Definition and Classification

Traumatic brain injury (TBI) is a widespread and complicated phenonmenon (Langlois Orman, Kraus, Zaloshnja, & Miller, 2011; Ponsford, 2013). A multitude of definitions of TBI and descriptions of sequelae have been provided over the years. Historical accounts of TBI (see McCory & Berkovic, 2001; Sablas, 2001) are consistent with current definitions and include closed and penetrating brain injuries, an altered state of consciousness (ASC), a loss of consciousness, (LOC), post-traumatic amnesia (PTA), other injury-related experiences, and neurobehavioural changes postinjury (e.g., Phineas Gage – Harlow, 1848/1868 [1999]). These presentations form the pillars for the current diagnostic criteria of the spectrum of TBI severity. Although there are multiple factors and grading systems to classify and diagnose TBI, current best-practice approaches use similar, but multidimensional criteria.

According to the American Congress of Rehabilitation Medicine Demographics and Clinical Assessment Working Group of the International and Interagency Initiative toward Common Data Elements for Research on Traumatic Brain Injury and Psychological Health (ACRM; Menon, Schwab, Wright, & Maas, 2010), a “TBI is

defined as an alteration in brain function, or other evidence of brain pathology, caused by an external force" (p.1637). Traditionally, TBI is classified by clinical assessment guidelines that include the duration of LOC/ASC, the duration of PTA (Crovitz & Daniel, 1987; Russell & Smith, 1961), assessment of visual responses, verbal responses, and motor function via the Glascow Coma Scale (GCS; Teasdale & Jennett, 1974), and the presence or absence of brain abnormalities via neuroradiological evidence.

The classification of injury severity is considered on a continuum (Alexander, 1995; Iverson & Lange, 2009). Traditional classification of injury severity ranges from mild, moderate, to severe TBI and is often based on GCS scores (13-15 mild; 9-12 moderate; 3-8 severe; Teasdale & Jennett, 1974) or length of PTA in acute care settings. Although Glascow Coma Scale scores and duration of PTA may hold some prognostic value in physical and cognitive outcome, respectively, for persons with moderate or severe TBI, these indices are especially not sensitive in determining the outcome in mild, albeit head trauma (e.g., Gomez, Lobato, Ortega, & de la Cruz, 1996; King, 1997; Lieh-Lai et al., 1992; Ponsford, Cameron, Fitzgerald, Grant, & Mikocka-Walus, 2011; Ponsford et al., 2000; Ruff, 1999). For example, a GCS score of 15 may be obtained by a person with no head trauma or by a person who has experienced 'mild' injury (Giza & Hovda, 2001). Similarly, loss of consciousness (LOC) is not a required criterion for classification of mild TBI (i.e., Kay et al., 1993) and has shown variable (to nil) prognostic value in predicting outcome on cognitive measures (e.g., Lovell, Iverson, Collins, McKeag, & Maroon, 1999).

Furthermore, GCS scores and other information may not be obtainable because the majority of mild head trauma goes unreported because many do not seek medical

treatment for their injuries (e.g., Sosin, Sniezek, & Thurman, 1996). As well, traditional neuroimaging via computerized tomography (CT) in mild head trauma typically does not demonstrate structural abnormalities (Bouida et al., 2013; Belanger et al., 2007; Bigler, 1999; Iverson, Lovell, Smith, & Frazen, 2000). For example, recently Bouida and colleagues (2013) conducted a prospective, multicenter study and found that only 13.8% (*n* = 218 of 1,582) of patients with mild/minor head trauma in emergency medical settings had positive CT findings. However, metabolic changes and/or microscopic changes such as the integrity of white matter pathways is not typically observed in traditional neuroimaging techniques (e.g., CT), but the neuropathophysiological changes in mild head trauma can be captured by newer techniques (Bigler, 2013) such as gradient echo magnetic resonance imaging (MRI), diffusion tensor imaging (DTI) or diffusional kurtosis imaging (DKI). Microhemorrages have been shown with gradient echo T2* MRI in an individual following a sports-related mild head trauma (Asif, Harmon, Drezner, & O'Kane, 2010) and this discovery is consistent with shear injury. As well, measures of DKI and DTI (i.e., fractional anisotrophy, mean diffusivity) have shown significant associations with cognitive outcome on neuropsychological measures for persons with mild (e.g., Grossman et al., 2013) or moderate/severe TBI (e.g., Sugiyama et al., 2009). Modern imaging techniques such as DTI and MRI are showing increasing sensitivity to detecting structural abnormalities and these techniques complement clinical observations for a multidimensional approach to assessment and diagnosis of TBI (see Belanger, Vanderploeg, Curtiss, & Warden, 2007 for review; Bigler, 2013; Bigler & Maxwell, 2012).

A plethora of definitions and grading systems of mild TBI has plagued the scientific and clinical communities. However, the current accepted best-practice definitions for mild TBI provide multidimensional and more uniform criteria (Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine [ACRM; Kay et al., 1993]; World Health Organization (WHO) Collaborating Task Force on Mild Traumatic Brain Injury [Carroll, Cassidy, Holm, Kraus, & Coronado, 2004]; and, the Center for Disease Control (CDC) Working Group [National Center for Injury Prevention and Control, 2003]). Kay et al. (1993; Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the ACRM) characterized mild TBI as a "traumatically induced physiological disruption of brain function" and outlined the following four criteria of which "at least one must be present: (1) any period of loss of consciousness, (2) any loss of memory for events before or after the event, (3) any alteration in mental state at the time of the injury (e.g., feeling dazed, disoriented, or confused), (4) focal neurological deficits that may or may not be transient" (pp. 86-87). The exclusion criteria include "(1) a loss of consciousness exceeding 30 minutes, (2) a GCS score below 13, and (3) PTA persisting longer than 24 hours" (p. 86-87) [each of which are indicators of more significant injury severity]. Over the years various terms have been used synonymously with mild TBI such as concussion, minor head injury, mild head injury, complicated mild TBI, uncomplicated mild TBI, and so forth (see King, 1997; Iverson & Lange, 2009). Mild head injury (MHI) is used throughout this dissertation with respect to our sample(s) to acknowledge the self-report of the injury and was adopted from the widely accepted definitions provided by Kay and colleagues (1993;

Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the ACRM) and the World Health Organization (WHO) Collaborating Centre for Neurotrauma Task Force on Mild TBI (Holm, Cassidy, Carroll, & Borg, 2005).

Lastly, many controversies exist regarding the classification and grading of severity of TBI (see Slobounov 2008 for a historical account; Wrightson, 2000). A categorical, rather than dimensional definition of TBI has often been adopted. Although TBI severity follows a continuum of disruption, ranging from very mild [minor], uncomplicated mild, complicated mild, moderate, severe, to catastrophic injuries that result in severe disability or death (Alexander, 1995; King, 1997; Iverson & Lange, 2009), this spectrum is often conceptualized as categorical rather than a dimensional aspect of injury severity (e.g., see Bodin, Yeates, & Klamar, 2012 for discussion). The categories of mild, moderate, and severe TBI have been considered ordinal and defining the cut-off criteria or boundaries has been challenging especially with respect to mild TBI (e.g., Kay, Newman, Cavallo, Ezrachi, & Resnick, 1992; King, 1997; Slobounov, 2008). Indicators of injury severity such as altered mental state, LOC, amnesia, neuroradiological evidence or neurological deficit are used in 'mild' head injury (Kay et al., 1993) to further define this classification into grades of injury severity. Grading systems of mild head trauma or concussion abound but many are similar. Cantu's (2001) grading scheme is one of the most common and is empirically-based: Grade 1/'mild' concussion – no LOC, may have experienced brief PTA or other postconcussion symptoms; Grade 2/'moderate' concussion – brief LOC, PTA not greater than 24 hours, and postconcussive symptoms experienced for up to 7 days; and, Grade 3/'severe' concussion – LOC longer than 1 minute, PTA greater

than 1 day, and experience of postconcussive symptoms for longer than 7 days (Cantu, 2001). Notably, 'mild' terminology does not suggest insignificant injury or transient injury (DeCuypere & Klimo, 2012; McMahon et al., 2014), nor does the term mild/minor head injury discount injury to the brain (e.g., Kay et al., 1992). Many behavioural, physical, cognitive, and affective difficulties after a 'mild' head trauma may present in a fashion similar to that of moderate or severe injury and only differ in the magnitude of the presentation (e.g., see Baker & Good, 2014; van Noordt, Dzyundzyak, Baker, Chiappetta, DeBono, & Good, 2014), which illustrates a continuum of injury severity.

Epidemiology

TBI is one of the most common health care problems worldwide and is a leading cause of death and disability (WHO - Neurological Disorders Report, 2006; Teasell, Aubut, Bayley, & Cullen, 2013 - Evidence Based Review of Moderate to Severe Acquired Brain Injury [ERABI]). TBI imposes a great impact on survivors, families, public health, and society with best estimates of 10 million people affected annually worldwide (see Hyder, Wunderlich, Puvanachandra, Gururaj, & Kobusingye, 2006 for review). Although the incidence of TBI varies amongst epidemiological studies due to differences in the operational definition of TBI or other research methodology, and much of the published estimates are often dated (e.g., Kraus & Nourjah, 1988; Canadian Institute of Health Research [CIHR], 2006), it is estimated that, on average, the global annual incidence rates range from 150 to 300 per 100,000 population per year in Western developed countries (see Tagliaferri, Compagnone, Korsic, Servadei, & Kraus, 2006; Kraus & Chu, 2005; Canadian Institute for Health Information [CIHI], 2007). The WHO states that children, young adults, and the elderly show the highest incidence rate with

injuries resulting primarily from motor vehicle collisions (MVC), falls, or violence-related injuries (WHO – Neurological Disorders Report, 2006; Teasell et al., 2013). Further, TBI is the leading cause of death and disability in children and youth. Also, males are 2 to 3 times more likely to sustain a TBI than are females (WHO, 2006; Kraus & Nourjah, 1988; Rutland-Brown, Langlois, Thomas, & Xi, 2006; Langlois, Rutland-Brown, & Thomas, 2004).

The majority of North American incidence rates have focused on the US population. Approximately 1.5 million TBIs are incurred annually in the United States (Langlois, Rutland-Brown, & Thomas, 2004; Thurman, Alverson, Dunn, Guerrero, & Sniezek, 1999). Langlois et al. (2004) estimated from data collected from the period of 1995 to 2001 that there were 1.1 million annual emergency department visits associated with TBIs, 235,000 hospitalizations associated with TBI annually, and 50,000 deaths as result of TBI. In this population-based study, falls accounted for 28.5% of TBIs (n = 398,000), motor vehicle collisions – 20% (n = 280,000), assault-related injuries – 11.2% (n = 156,000), and TBI of other/unknown causes – 40.3% (n = 562,000) (Langlois et al., 2004). The etiology of TBI also varies across age groups. For example, Langlois et al. demonstrated that falls were the most common cause of TBI for infants and children up to 4 years of age as well as for those 75 years and older, whereas MVCs and assaults were the leading causes of TBIs in adolescents from 15-19 years of age (Langlois et al., 2004; Center for Disease Control [CDC], 2010).

There is limited research on the epidemiology of TBI in Canada and most estimates are based on US statistics; however, the incidence of TBI follows a similar pattern. According to the Brain Injury Association of Canada (BIAC) an estimated 1.4 million

Canadians are living with the effects of TBI. The CIHR report on Head Injuries in Canada (2006) reported that in 2003-2004 there were 16,811 emergency department hospital admissions for traumatic head injuries which accounted for 9% of all trauma admissions. Across all age groups, falls were the leading cause of TBIs (45.5%; *n* = 7637), followed by motor vehicle collisions (36%; *n* = 5,970), other causes (10%; *n* = 1,734), and purposeful injuries such as assaults (8.5%, *n* = 1,470). Notably, 28% of children and youth were admitted to the hospital because of a sports-related TBI, and, in contrast, only 8% of adult TBIs were related to sports or recreational activities (CIHI, 2006). Impressively, over a decade (1993/1994 to 2003/2004) the incidence of TBI has been significantly reduced for ages 0-19 years (53% i.e., 10,589 in 1994-1995 to 4,966 in 2003-2004) and 20-59 years, but there has been a 4% increase of TBIs for those over 60 years of age (*n* = 4,882 in 1994- 1995 and 4,902 in 2003-2004). Furthermore, the number of TBI-related deaths has been substantially reduced for all age groups (CIHR, 2006). These data suggest that progress is being made in the prevention of TBI in Canada. Similar improvement regarding mortality due to TBI has been made in the USA (see DeCuypere & Klimo, 2012 for a discussion).

Incidence rates also vary as a function of severity of injury and method of report. A Canadian study by Zygun et al. (2005) estimated the annual incidence rate of severe TBI to be 11.4 per 100,000. At the other end of the continuum of injury severity, mild TBIs account for approximately 75 to 90% of all TBIs (CIHI, 2007; Kraus & Nourjah, 1988; Kraus & Chu, 2005; Ryu, Feinstein, Colantonio, Streiner & Dawson, 2009). For example, Ryu et al. (2009) calculated the incidence rates for hospital-treated mild TBI in Canada to be approximately 426 - 535 per 100,000. Ryu et al.'s estimates of incidence rates for mild

TBI increased to 653 per 100,000 when family physician-based treatment was included. Yet, much of mild head trauma is not reported and many do not seek medical treatment for their injuries or result in hospital admissions (e.g., 25% in Sosin et al., 1996; CIHR, 2006). Therefore the incidence of mild head trauma is greatly underestimated (Teasell et al., 2013) and increases when self-reported injuries are included (e.g., Sosin et al., 1996; Baker & Good, 2014).

In the United States, an estimated 131 - 367 per 100,000 mild TBIs occur per year (Kraus et al., 1984). However, studies including self-reports of injury illustrate higher incidence. For instance, Sosin and colleagues (1996) estimated the head injury incidence in the US to be 618 per 100,000 using self-reported interview data from a nationally representative civilian sample (US Census Bureau Injury Supplement 1991; N = 120, 032). Head injury in this survey study was defined as trauma with an associated loss of consciousness occurring in the past 12 months that did not result in death or need for long-term residential care. This definition likely captured both mild and moderate head injuries. Sosin et al. found that over 25% of this sample did not receive medical treatment for their TBI. Of the 75% of persons that reportedly sought medical care, 14% were treated in outpatient clinics or physician offices, 35% were treated in the emergency department, and 25% were admitted to hospital due to the TBI (Sosin et al., 1996) – with the latter indicating greater injury severity.

These findings (i.e., Sosin et al., 1996) are similar to a more recent population-based study in New Zealand by Feigin et al. (2013) that used both prospective and retrospective methods to obtain TBI event history. Feigin et al. estimated the incidence of TBI to be approximately 790 cases per 100,000 (including all ages and all severity

classifications) from data collected across 2010-2011 in a large population based study. These rates also differed as a function of severity of injury classification such that mild TBI was estimated to be 749 cases per 100,000 and moderate to severe cases were 41 per 100,000. Notably, the risk for incurring a mild TBI was approximately 18 times greater than the risk for sustaining a moderate to severe TBI (Feigin et al., 2013). Feigin and colleague's case ascertainment method accounted for individuals with mild TBI who were not admitted to the hospital which is important because oftentimes these persons are excluded from hospital-based epidemiological studies of TBI incidence. This latter point may account for the increased incidence reported in this study relative to other studies (e.g., Sosin et al., 1996).

Accurate, robust, and up-to-date information about the prevalence of TBI does not exist for the Canadian population, but data such as that presented by Feigin and colleagues (2013) suggest that the current prevalence in other Westernized, high-income countries may be far greater than prior estimates. Furthermore, the incidence of mild TBI reported by Feigin and colleagues is similar, albeit elevated, to that of other studies that include self-report methods of head trauma history (e.g., Segalowitz & Lawson, 1995; Sosin et al., 1996; Vanderploeg, Curtiss, Luis, & Salazar, 2007), which captured those who may not have sought medical treatment for their injury. Lastly, the estimated and outdated incidence of TBI worldwide warrants an urgent need for reliable community and public health surveillance systems to improve the monitoring and evaluating of the incidence, etiology, and outcomes of TBI. Incidence data should also include record of head traumas that do not result in hospital admission as well as those that received

medical treatment for their injury to effectively inform prevention strategies, as well as treatment strategies, and other services for persons with TBI.

Mechanisms of Traumatic Brain Injury

TBIs are heterogeneous with variable presentations; however, a commonality is that trauma to the head results from external forces be it from closed head injuries in the form of blunt trauma and/or inertial forces (i.e., acceleration – deceleration; rotational forces), or penetrating injuries (e.g., McAllister, 2008; Ponsford, 2013). The etiology and pattern of these biomechanical events plays a role in the extent of tissue damage (McAllister, 2011). A detailed discussion of the biomechanics and neuropathological processes is beyond the scope of this dissertation, but key aspects are highlighted in the following discussion (for review see Farkas & Povlishock, 2007; Namjoshi et al., 2013; Ponsford, 2013; Prins, Greco, Alexander & Giza, 2013).

TBI is associated with both focal and diffuse injury (see DeCuypere & Klimo, 2012; Katz, Zasler, & Zafonte, 2007; Ponsford, 2013). Diffuse injury involves axonal injury (i.e., diffuse axonal injury [DAI]) as a result of sudden acceleration-deceleration or rotational forces that exert damage to the axons. Diffuse axonal injury can have widespread impact on the brain and behaviour due to disrupted axonal communication (e.g., Sugiyama et al., 2009; Wilde et al., 2005). Focal injuries, on the other hand, refer to the occurrence of contusions, haematomas, and lacerations and may occur as result of a coup-contrecoup injury in which the brain smashes against the skull opposite to the side of impact (see Drew & Drew, 2004; Meaney & Smith, 2011). Focal injuries tend to occur at the site of impact/high-energy force (coup) or contrecoup area and evidence neurological deficits related to a more restricted/focal area of the brain (DeCuypere &

Klimo, 2012; McAllister, 2011; Ponsford, 2013). Dynamic mechanical forces can occur with or without direct external trauma to the head (Varney & Varney, 1995) and act on the skull and brain which results in linear or rotational movement of head and skull (Barth, Freeman, Boshek, & Varney, 2001; Greaves et al., 2009; Meaney & Smith, 2011; Varney & Varney, 1995). This movement of the brain against the irregular bony surface at the base of the skull and the frontal/anterior and temporal/middle fossae is especially damaging to the tissue (Drew & Drew, 2004; Gaetz, 2004; Katz et al., 2007; McAllister, 2011; Wilde et al., 2005). The ventromedial prefrontal cortex (VMPFC) and the temporopolar regions are particularly vulnerable to disruption due to their close proximity to the bony protusions of the skull (e.g., Gentry, Godersky, & Thompson, 1988; Lezak et al., 2012; McAllister, 2011; Morales, Diaz-Daza, Hlatky, & Hayman, 2007). Many trauma injuries follow an anterior-posterior or a posterior-anterior trajectory (e.g., as in a motor vehicle collision) which puts the frontal and occipital regions at risk for injury based on their location (DeCuypere & Klimo, 2012; Gentry et al., 1988; McAllister, 2008; 2011).

Acceleration-deceleration and/or rotational forces exert maximal forces on neurons (primarily the axons) and blood vessels, which result in strain and shear injuries (Gennarelli et al., 1982; Misra & Chakravart, 1984; McAllister, 2011; Varney & Varney, 1995). White matter tracts are susceptible to shear injury (see Bigler, 2013; Sugiyama et al., 2009) and the white-gray matter junctions are especially at risk including the corpus callosum, fibre tracts in the frontal and temporal regions, the basal ganglia, the periventricular region, as well as the cerebellar peduncles (Bigler, 2013; Gaetz, 2004).

Shearing injuries to blood vessels are also common and result in micro or macrohemmorhages (see Asif et al., 2010; Gennarelli & Graham, 1998).

An alternative way to describe the biomechanical and pathobiological processes that occur during TBI is in terms of the primary or secondary aspects of the injury (rather than focal or diffuse injury) (Gaetz, 2004; Ponsford, 2013). Many injuries result from a combination of both focal and diffuse as well as primary or secondary injury events (McAllister, 2008). Primary injury refers to damage from the direct contact to the head and/or inertial forces that disrupt the brain parenchyma (Ponsford, 2013; Prins et al., 2013), whereas secondary injury may be characterized by ongoing cellular events that comprise the complexity of the 'biochemical cascade' (Giza & Hovda, 2001; Giza & Hovda, 2004) that occurs following trauma to the head as well as other secondary responses such as changes in cerebral blood flow and inflammation (Ponsford, 2013). A variety of established experimental animal models are available that elucidate secondary injury processes in TBI (e.g., Generalli & Graham, 1998; Farkas & Povlishock, 2007) especially models that mimic closed head injury such as that incurred via motor vehicle collisions or sports-related injuries such as a concussion (e.g. Giza & Hovda, 2001; 2004; Farkas & Povlishock, 2007). The research has demonstrated that even 'milder' injuries such as concussion are subject to neuropathological changes and cerebral dysfunction (Bigler, 2013; Giza & Hovda, 2004; also see Len & Neary, 2011).

The multifaceted biochemical processes that occur following closed head injury affect brain function (Giza & Hovda, 2001; 2004; Prins et al., 2013). Data from fluid percussion models of mild damage (i.e., primarily Giza & Hovda e.g., 2001) demonstrated that neuronal disruption results in a deregulated flux of ions, metabolic

changes, and a mismatch of energy resources that ultimately result in alterations in cerebral blood flow. In general, when an impact/force smashes the brain against the skull (as in concussion), there is a massive efflux of potassium and an influx of calcium. These ionic changes result in an increased release of the excitatory neurotransmitter glutamate. Glutamatergic activity at the N-methyl-D-aspartate receptors precipitates further depolarization of neurons and an influx of calcium – the latter affects the mitochondria, and, as a consequence, results in neuronal suppression and accompanying hypometabolism of glucose. To restore the ionic balance, there is increased activity of the sodium-potassium pumps which increases the use of glucose and depletes energy stores – creating what has been referred to as a 'cellular energy crisis' (Giza & Hovda, 2001; Prins et al., 2013). The metabolizing of glucose results in an accumulation of lactate which is implicated in intracellular dysfunction. Furthermore, disruption to the axonal membranes causes mechanical breakage of microtubules, which results in impaired axonal transport. As well, the shearing of endothelial cells in small blood vessels impairs the regulation of the blood-brain barrier as well as alters cerebral blood flow and may result in focal ischemia (Barkhoudarian, Hovda, & Giza, 2011; Blennow et al., 2012; Giza & Hovda, 2001; 2004). Data from humans have also demonstrated decreased cerebral blood flow after mild to severe TBI (e.g., see Arvigo et al., 1985; Taylor & Bell, 1966). Alterations in other indices of vascular function such as heart rate variability have also been documented postinjury. For example, Gall, Parkhouse, and Goodman (2004) examined heart rate variability between athletes (hockey players) who had recently sustained a concussion (~1.8 days postinjury) and persons without head trauma and found no significant differences between these groups in this parameter at rest. However, when

they engaged in an exercise task that posed a physical challenge the athletes who had sustained a concussion demonstrated significantly lower heart rate variability relative to controls (Gall et al., 2004).

The mechanisms that result in brain tissue disruption and damage are complex and involve a variety of neuropathophysiological processes (e.g., for review see Farkas & Gaetz, 2004; Povlishock, 2007; Ponsford, 2013; Prins et al., 2013). The neurobehavioural presentation after TBI and associated challenges is multifarious; it is important to understand the biomechanisms involved in the injury event, the etiology of the injury, the regions of the brain that are at particular risk for disruption, and the multifaceted pathophysiological changes that occur during and following injury to the head/brain. Understanding the occurrence of primary injury and the evolution of secondary damage provides a better understanding of the clinical profile of persons with TBI (Bigler, 2013; Gaetz, 2004; Giza & Hovda, 2004). Furthermore, although persons with trauma to the head have differing degrees of injury severity and may have different neurobehavioural profiles, there are common biochemical processes that occur regardless of the severity of trauma (Len & Neary, 2011; Prins et al., 2013).

Sequelae following Traumatic Brain Injury

The multifaceted pathophysiological changes after TBI result in neurological and neurobehavioural challenges (McAllister, 2011). Moreover, many complex medical and psychiatric issues may complicate the interpretation and presentation of neurobehavioural sequelae postinjury (Eslinger, Zappala, Chakara, & Barrett, 2007; Ponsford et al., 2000). The sequelae after TBI include changes in physical, cognitive, emotional, and social functioning (e.g., Bornhofen & McDonald, 2008; Ietswaart et al., 2008; Naqvi et al.,

2004; Sloan & Ponsford, 2013; Stuss & Levine, 2002) – all of which present significant challenges to recovery and reintegration (e.g., Draper, Ponsford, & Schönberger, 2007; Hammond, Davis, Whiteside, Philbrick, & Hirsch, 2001; Larsson et al., 2013) and will be discussed further in the current research.

Cognitive challenges. As previously discussed, the prefrontal and frontal areas are particularly vulnerable in TBI (e.g., Gentry et al., 1988; McAllister, 2011; DeCuypere & Klimo, 2012). Disruption to these regions is associated with hallmark sequelae of TBI including attentional and memory problems, reduced processing speed, and impaired executive functioning (e.g., Lezak et al., 2012; Raskin, Mateer, & Tweeten, 1998; Shallice & Gillingham, 2013; Stuss & Levine, 2002). Neuropsychological assessment is particularly useful in providing standardized and objective indices of functioning postinjury in these domains (and others) (Lezak et al., 2012). For example, Sarno, Erasmus, Lipp, and Schlaaegel (2003) demonstrated deficits in reaction time across modalities (i.e., visual, auditory, and tactile stimuli) for persons with moderate to severe TBI. Notably, simple reaction time tasks may result in similar speeds of performance for persons with and without TBI; however, as the complexity of the task increases, the reaction time often illustrates impairments in speed of processing for persons with TBI (e.g., Stuss, Stethern, Hugenholtz, Picto, Pivik, & Richard, 1989; Stuss & Levine, 2002). In addition, the consistency of slowed processing speed and continued cognitive challenges across the recovery period has been documented (e.g., Salmond, Menon, Chatfield, Pickard, & Sahakian, 2006; Stuss & Levine, 2002). For instance, Salmond and colleagues (2006) demonstrated that at both 6 months and 3 years postinjury persons with

moderate to severe TBI demonstrated cognitive impairments in attention, memory (paired associates task), and reaction time relative to noninjured controls.

Executive functioning is also commonly compromised following disruption to the frontal lobes (Constantinidou, Wertheimer, Tsanadis, Evans, & Paul, 2012; Stuss & Levine, 2002). Executive functioning may be broadly defined as a complex group of functions that permit integrative, higher-order processes such as metacognition, attention, inhibition, working memory, monitoring, task setting/organization, planning, problem-solving, and cognitive control (Constantinidou et al., 2012; Elliot, 2003; Stuss, 2011; Stuss & Levine, 2002). Stuss (2011) argues that these functions are domain general processes that work in concert to establish cognitive control and that there is no single 'executive' (see also Shallice & Gillingham, 2013). In terms of disinhibition, cognitive flexibility and decision-making, it is well documented that persons with damage to the PFC show impairments in judgment and decision-making and exhibit disinhibited behaviours (e.g., Phineas Gage – Harlow, 1848; patient EVR – Eslinger & Damasio, 1985). Furthermore, persons with damage to the VMPFC/OFC have challenges regulating behaviour in response to feedback (e.g., Roberts et al., 2004; Naqvi et al., 2004), which may similarly present as difficulties in adapting to or changing their behaviour based on the actions of another individual during social interactions (e.g., Kuhn et al., 2010). These behaviours pose a challenge for successful social reintegration (Beer, Heerey, Keltner, Scabini, & Knight, 2003; Beer, John, Scabini, & Knight, 2006; Eslinger & Damasio, 1985; Kuhn et al., 2010).

Emotional challenges. With respect to emotional functioning difficulties, persons with disruption to the frontal lobes have demonstrated hemispheric asymmetry in terms

of emotional functioning impairments (e.g., damage [or lesion] to right or left prefrontal cortex). Generally, persons with damage to the right PFC present with anosognosia (e.g., Lezak et al., 2012), a 'rose-coloured' view of self, and perhaps one's circumstances also termed 'la belle indifference' (Pierre Janet [1849-1947]; see Iezzi, Duckworth, & Adams, 2004; Martelli, Nicholson, & Zasler, 2007). In a similar fashion, persons with disruption involving the VMPFC region often present with flattened affect and attenuated physiological responsivity to stimuli (e.g., Damasio, Tranel, & Damasio, 1990; Naqvi, Shiv, & Bechara, 2004; Roberts et al., 2004; Tranel & Damasio, 1994). The VMPFC region is of particular interest given its extensive connectivity with limbic regions and its involvement in the modulation of emotional responses (e.g., Barbas et al., 2003; Hansel & von Kanel, 2008; Wallis, 2007) as well as the perception of self and others (Beer et al., 2003; 2006; Beer, 2007; Beer & Hughes, 2010; Beer, Lombardo, & Bhanji, 2010). Persons with lesions to the orbitofrontal cortex (OFC) have been found to hold unrealistic positive self views (e.g., see Beer et al., 2006; 2007) therefore it has been suggested that this region, like the right PFC, is involved in a positivity bias or '*rose-coloured glasses*' perspective (Beer & Hughes, 2010; Beer et al., 2010). On the other hand, disruption to the left PFC often results in emotional lability and dyscontrol (e.g., emotional outbursts – see Woolley et al., 2004; Hale & Fiorelli, 2004). The view of PFC asymmetry in emotional functioning (i.e., valence) that has been derived from lesion studies has been corroborated in research with healthy individuals (e.g., see Davidson, Jackson, & Kalin, 2000 for discussion). However, oftentimes the disruption to the brain is not focal and the damage is diffuse which can result in a complex behavioural presentation.

Furthermore, impairments in emotion recognition (e.g., Ietswaart et al., 2008; Bornhofen & McDonald, 2008), altered responsivity to emotional stimuli (Hopkins et al., 2002; Hornak et al., 1996; McLelland & McKinlay, 2013), and altered socioemotional functioning after moderate to severe TBI may pose a barrier to successful psychosocial functioning (e.g., DeSousa et al., 2010; Milders et al., 2003; Ponsford & Schöenberger, 2010). TBI commonly impairs both the expression of and experience of emotions. Research has demonstrated that persons with moderate to severe TBI experience attenuated emotional reactivity, particularly to negatively-valenced stimuli, in terms of both subjective ratings and sympathetic indices of emotional arousal such as electrodermal activation and the startle reflex (see Angrilli, Palomba, Cantagallo, Maietti, & Stegnano, 1999; Dethier, Blairy, Rosenberg, & McDonald, 2013; Hopkins et al., 2002; McDonald et al., 2011; Roberts et al., 2004). For example, Hopkins et al. (2002) found that persons with moderate to severe closed head injury demonstrated a lessened ability to identify emotional facial expressions and produced altered responsivity to emotional stimuli relative to age- and sex-matched controls. Electrodermal activation (EDA), an index of emotional arousal, was found to be significantly attenuated for persons with closed head injury when compared to the control group, but only for negatively valenced facial expressions. This illustration of emotional hyporesponsiveness was discussed by Hopkins et al. to be consistent with the data of persons with frontal lesions, especially those with disruption to the orbital and medial regions of the PFC (OMPFC), who show differential responsivity to socially relevant stimuli (i.e., see Damasio et al., 1990). The OMPFC has been implicated in the modulation of emotional arousal via involvement with the sympathetic and parasympathetic systems as well as its connections with the

amygdala (Barbas, Saha, Remple-Clower, & Ghashghaei, 2003; Barbas, 2007; Ghashghaei, Hilgetag, & Barbas, 2007). Although neuroimaging evidence was not available in the study by Hopkins et al. (2002) to corroborate the location of the closed head injury, nonetheless, the findings demonstrated that persons with moderate to severe TBI evidence challenges in affect recognition and alterations in physiological responses to negative facial expressions – both suggest altered OMPFC functioning.

Similarly, Spikman et al. (2013) demonstrated that persons with moderate to severe TBI were significantly impaired on an emotional recognition task relative to healthy controls especially with respect to recognizing facial expressions of negative emotions of fear, anger, sadness, and disgust. Moreover, both persons with TBI and their significant others (proxy) reported significantly more behavioural problems on the Dysexecutive Questionnaire (DEX; component of the Behavioral Assessment of the Dysexecutive Syndrome) than did noninjured controls. Spikman et al. also found that impaired emotion recognition abilities were significantly related to the ratings of behavioural problems provided by their significant others, but not to the self-reported ratings provided by those with TBI. This finding suggests a decreased awareness of behavioural challenges for persons with TBI (i.e., anosagnosia). The results of the study by Spikman et al. (2013) demonstrate the importance of socioemotional signals, such as that gleaned from facial expressions of emotions, and its relation to behavioural challenges (Spikman et al., 2013; also see Khun et al., 2010). As well, persons with moderate to severe TBI may lack insight into their socioemotional impairments (Spikman et al., 2013).

It has also been suggested that persons with TBI may also have decreased empathic experiences relative to persons who have not experienced TBI (e.g., de Sousa et al., 2011;

Williams & Wood, 2010). Along these lines, persons with TBI have demonstrated impairments in imitating other person's facial expressions of negative emotions such as anger (see de Sousa et al., 2011). It is likely that these (and other) behavioural changes often impact social interactions as a result of inappropriate socioemotional functioning (e.g., emotional indifference, aggressive interactions e.g., in Iezzi, et al., 2004; Martelli et al., 2007; decreased appreciation/understanding of humour – e.g., Shammi & Stuss, 1999). Such emotional challenges have also been associated with disruption to the ventromedial PFC, which is implicated in the modulation of autonomic responsivity (Bornhofen & McDonald, 2008; Naqvi et al., 2004; see Wallis, 2007).

In addition, another aspect of emotional functioning is stress responsivity. There is evidence to suggest that persons with moderate to severe TBI experience more life stressors (e.g., Dawson et al., 2004). However, examinations of persons with head trauma in terms of their physiological and self-reported indices of responses to stress in both the environment and laboratory setting are, at best, limited (e.g., Bay, Sikorskii, & Gao, 2009; Covassin & Bay, 2012; Hanna-Pladdy, Berry, Bennett, Phillips, & Gouvier, 2001; Moore, Terryberry-Sphor, & Hope, 2006), and demonstrate variable responses. Although given the reduced self-reported and physiological responsivity to emotional stimuli of the aforementioned studies (e.g., Bornhofen & McDonald, 2008; Hopkins et al., 2002; Spikman et al., 2013), it is likely that persons with head trauma may have reduced responses to stressors and/or an altered [overly positive] perception of these experiences (or one's experiences e.g., Beer et al., 2003; 2006; 2010; Beer & Hughes, 2010) - both of these factors may play a role in differential responses to stressors.

Lastly, individual differences such as sex (e.g., McCormick, 2007; see Kudielka & Kirschbaum, 2005 for review; Kudielka, Hellhammer, & Wust, 2009) or preinjury characteristics (e.g., personality) may also influence emotional responsivity and/or stress responses postinjury; however, limited research has examined these factors in the TBI population, let alone in the context of responses to stressors (e.g., Covassin & Bay, 2012; Bryant, 2011). The findings of sex differences in both physiological and self-reported responses to laboratory and experiential stressors for persons without neurological compromise have been variable (e.g., see Kudielka, Hellhammer, & Wust, 2009). For example, men have shown an increased salivary cortisol response to a psychosocial stressor relative to women (Kirschbaum, Wust, & Hellhammer, 1992), whereas other research has shown that women perceive stressful life events such as living in an unsafe area or experiencing personal injury or illness as more distressing than males (e.g., Carballo & Carbena, 1997). To our knowledge, sex differences in stress responses have been rarely examined for persons with TBI (see Covassin & Bay, 2012). One study by Covassin and colleagues (2012) did not demonstrate any significant difference in their perceived experience of chronic stress between men and women with mild to moderate TBI. Covassin and Bay (2012) found there was no significant difference between women and men with TBI for their ratings of the extent to which they perceived their experiences in the past month to be unpredictable, lacking control, and/or difficult to manage. The research in this area is scarce and is likely because most of the attention regarding stress responses for persons with TBI has been on post-traumatic stress disorder (PTSD) after TBI (e.g., Bryant, 2011; Bryant & Harvey, 1999; Rogers & Read, 2007)

Preinjury characteristics such as personality are another factor that may influence emotional functioning. Some research (e.g., Hibbard et al., 2000) has shown that approximately 25% of persons who had sustained a moderate to severe TBI had been diagnosed with a personality disorder prior to their injury. Antisocial Personality Disorder as well as Obsessive Compulsive Personality Disorder were among the most frequent preinjury diagnoses (Hibbard et al., 2000). These data (and others) may suggest that persons with certain personality characteristics such as aggressiveness or risk-taking may be at particular risk for experiencing a TBI (see Kraus & McArthur, 2006 for discussion), but prospective studies are needed. As well, changes in personality postinjury are frequently observed (Hibbard et al., 2000; Obonsawin et al., 2007) and are typically long-lasting (i.e., 15 years postinjury– Oddy, Coughlan, Tyerman & Jenkins, 1985). Approximately 55% of persons with TBI may have an acquired personality disorder diagnosis (Hibbard et al., 2000). Among the acquired personality disturbances postinjury is a profile of 'acquired sociopathy' (Saver & Damasio, 1991) which is consistent with the personality characteristics of psychopathy (Hare, 1993). Acquired sociopathy has been observed in persons with frontal lobe lesions (especially those with disruption to the VMPFC) in that persons with frontal lobe disruption demonstrate aberrant social behaviours and flat affect/underarousal (e.g., Barrash, Tranel, & Anderson, 2000; Blair & Cipolotti, 2000; Damasio et al., 1990; Eslinger & Damsio, 1985; Saver & Damasio, 1991). Damasio and colleagues (1994; 1998) have proposed that this "acquired sociopathy" profile results from impairments in one's internal representations of body state (i.e., "somatic markers" such as autonomic activity) that are hypothesized to provide guidance in decision making and responsivity to socioemotional

stimuli. In summary, all of the aforementioned cognitive and emotional sequelae may pose challenges to successful psychosocial functioning and quality of life after TBI (e.g., Anderson, Brown, Newitt & Hoile, 2011; Petchprapai, & Winkelman, 2007).

Mild head trauma sequelae. Although there has been much research that has examined the effects of moderate and severe TBI, the sequelae after milder forms of head trauma (i.e., mild traumatic brain injury [MTBI]; mild head injury [MHI]) is not well understood and the persistence of cognitive and emotional difficulties is often debated (e.g., Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005; Bernstein, 1999; Carr, 2007; Chuah, Maybery, & Fox, 2004; Dikmen et al., 1986; Len & Neary, 2011; Levin et al., 1987; McCrea, 2008; Moore, Terryberry-Spohr, & Hope, 2006; Panayiotou, Jackson, & Crowe, 2010). Mild head trauma is often accompanied by symptomology such as experiencing headaches, disrupted vision, or changes in sleep; attentional, memory and executive functioning difficulties, and, alterations in mood (Carr, 2007; Iverson & Lange, 2009). Collectively these symptoms are often referred to as Post-Concussion Syndrome (PCS; Carr, 2007; Harvey & Bryant, 1998; Iverson & Lange, 2009; Levin et al., 1987; McCauley et al., 2007; World Health Organization – International Classification of Disease 10th Edition [ICD-10], 1992). These postconcussive symptoms oftentimes subside after approximately 3 months and preinjury functioning is suggested to resume (e.g. Dikmen et al., 1986; Levin et al., 1987). However, the Center for Disease Control and Prevention (2007) has reported that 10 to 15 % of individuals continue to experience persistent difficulties in a variety of domains – socioemotional, physical, psychological, behavioural, and vocational (e.g., Carr, 2007; Chen et al., 2008; Gouvier et al., 1992; Hanna-Pladdy et al., 2001; Hellawell et al., 1999; Hopkins et al., 2005; Raskin et al.,

1998). Other researchers (e.g., Iverson & Lange, 2009) have suggested that 5% may be a more realistic estimate of this "miserable minority" (Ruff, Camenzuli, & Mueller, 1996) and that higher estimates may reflect the fact that the constellation of postconcussion symptoms are not specific to mild head injury and are commonly reported by the general and healthy noninjured population (e.g., Gouvier, Uddo-Crane, & Brown, 1988; Iverson & Lange, 2003; Wong, Regennitter, & Barrios, 1994).

Regardless of the incidence, Dean and colleagues (2012) have shown differences in somatic and cognitive complaints between persons with mild TBI and noninjured controls such that persons with mild TBI reported significantly greater experience of headaches, dizziness, nausea, and taking longer to think about things (Dean, O'Neill, & Sterr, 2012). In another study, King and Kirwilliam (2011) demonstrated that increased PCS severity for persons with mild TBI was related to poorer, and measurable, neuropsychological and psychosocial outcomes (see also Stalnacke, 2007). These findings do not clarify the mechanisms that initiate and/or perpetuate PCS symptomology (see Silverberg & Iverson, 2011 discussion), but they do suggest that PCS-like symptoms are substantive and should not be disregarded as 'general' in nature. In a similar fashion, significant associations have been demonstrated between microstructural changes and severity of PCS symptomology (e.g., Smits et al., 2011). For example, Smits and colleagues (2011) conducted a study using DTI and found that white matter integrity (i.e., mean diffusivity measure) was negatively related to increased PCS severity. These findings (e.g., Smits et al., 2011) demonstrate evidence of neuropathological, rather than psychiatric (e.g., Mittenberg & Strauman, 2000), substrates of postconcussive symptoms.

Of particular interest, the research regarding alterations in emotional functioning following mild head trauma has been inconclusive. For example, a meta-analysis by Panayiotou et al. (2010) found small, or minimal, effects on emotional functioning after mild TBI. However, other research (e.g., see Moore et al., 2006; Rao et al., 2010) has shown significant changes in emotional functioning in terms of increased anxiety (Byrant & Harvey, 2000) or depressive episodes (e.g., Rao et al., 2010) following mild TBI. Research on physiological indices of emotional functioning of persons who have sustained mild head trauma has been extremely limited (e.g., Baker & Good, 2014; Bay, Sikorskii, & Gao, 2009). To explore this gap in the literature, we (e.g., St. Cyr [Baker] & Good, 2007; Jung & Good, 2007; Baker & Good, 2014) have examined indices of emotional functioning and stress responsivity in the mild head injury population.

Across several studies (described in Baker & Good, 2010; 2014) we have tried to determine whether persons with self-reported mild head injury (MHI; i.e., sufficient to produce an 'altered state of consciousness'; Kay et al., 1993), similar to persons with moderate or severe TBI, present with altered emotional arousal (i.e., hypo- or hyperarousal). Studies conducted by the Brock University Neuropsychology Cognitive Research Lab (e.g., Jung & Good, 2007; St. Cyr [Baker] & Good, 2007; Baker & Good, 2010; 2014) have examined the emotional arousal status of university students with self-reported MHI and its relation to cognitive performance. Notably, this research was based on the *Yerkes-Dodson Law* (Yerkes & Dodson, 1908), which posits that performance is enhanced by arousal until an optimum level is reached, then performance decreases again as arousal levels exceed optimum, forming the inverted-U shaped relationship (see Lupien, Maheu, Tu, Fiocco, & Schramek, 2007 for review).

When we began investigating the emotional arousal sequelae after mild head trauma (Jung & Good, 2007; St. Cyr [Baker] & Good, 2007), we originally expected that persons with MHI, like those with moderate and severe neural disruption (e.g., Dawson et al., 2004), would be particularly vulnerable to stress (e.g., Hanna-Pladdy et al., 2001), and would, therefore, have higher self-reports of stress or anxiety disorders (e.g., Dawson et al., 2004; Gouvier et al., 1992; Hanna-Pladdy et al., 2001; Harvey & Bryant, 1998; 2000; Kennedy et al., 2004). We hypothesized that students with self-reported MHI would be even more disadvantaged in terms of cognitive performance (i.e., exhibit verbal and visuospatial memory difficulties as in Chuah et al., 2004; Hopkins et al., 2005; Levin et al., 1987; Raskin et al., 1998) with heightened anxiety relative to their no-MHI counterparts, and would perform best on cognitive tasks when anxiety levels were low (i.e., Yerkes-Dodson relationship).

As expected, students with no history of head trauma (30% of sample) performed best when less anxious and recall accuracy on memory tasks (e.g., Rey Complex Figure, Osterreith, 1944; WMS-III, 1997) was poorer when more anxious (St. Cyr [Baker] & Good, 2007). Contrary to our hypotheses, students with self-reported MHI acknowledged less anxiety relative to students without MHI. Moreover, students with self-reported MHI were not disadvantaged in memory performance due to an increased anxiety response, such that individuals with MHI performed optimally when more anxious and performance declined when less anxious (St. Cyr [Baker] & Good, 2007).

In a concurrent study from our lab, Jung and Good (2007) found that relative to their no-MHI counterparts, students with self-reported MHI were less physiologically aroused in general (as measured by heart rate), despite reporting increased experience of life stressors such

as financial or relationship difficulties. In this study, arousal state was experimentally manipulated by a psychosocial stressor (modified Trier Social Stress Test [TSST]; Kirschbaum, Pirke, & Hellhammer, 1993). Similar to the findings of St. Cyr [Baker] and Good (2007), the results indicated that students with self-reported MHI performed better on an attentional task with heightened physiological arousal, than lower arousal; in contrast, students with no history of head trauma performed more poorly with heightened arousal, and performed better with lower/moderate arousal. Further, students with MHI were less responsive to the psychosocial stress manipulation than were students with no-MHI (Jung & Good, 2007).

The results of these two studies (Jung & Good, 2007; St. Cyr [Baker] & Good, 2007) suggested that university students with self-reported MHI were not hyperaroused as we had originally hypothesized, but rather demonstrated lower arousal (as indexed by anxiety measures and physiological measures [i.e., heart rate]) relative to their no-MHI cohort. Based on these findings, we questioned the models of hyper- or hypoaroused emotional sequelae after neural disruption and suggested that persons with even mild head trauma may present with an altered emotional arousal pattern characterized by *underarousal* that is similar to persons with moderate to severe disruption to the VMPFC (e.g., Bechara, 2000; Bechara, Damasio, & Damasio, 2000; Hopkins et al., 2002; Naqvi et al., 2004; Tranel & Damasio, 1994) despite increased reports of experiential stressors (e.g., Dawson et al., 2004). This discrepancy may also indicate an altered perception of emotional events (e.g., positivity bias – Beer et al., 2003; 2006).

We (Baker & Good, 2010; 2014) conducted another study to continue to examine the models of hypo- or hyper- emotional arousal in this population to replicate and extend our prior findings (Jung & Good, 2007; St. Cyr [Baker] & Good, 2007) as well as to

explore some unique aspects of emotional arousal, stress responsivity, neurocognitive performance, and postconcussive symptom reporting as a function of a history of self-reported mild head trauma. These variables were also examined in terms of severity of head injury (i.e., those with no-MHI, those with MHI and no loss of consciousness, and those with MHI who reported a loss of consciousness). We wanted to examine the influences of modifying arousal state not just by increasing/activating the system via a psychosocial stressor as in a previous study (i.e., Jung & Good, 2007), but also by using a decreasing/relaxing technique. We also examined whether severity of injury would follow a gradient continuum of underaroused status (e.g., Alexander, 1995; Iverson & Lange, 2009). Neurocognitive performance of persons with and without MHI in more detail, especially executive functioning skills (e.g., cognitive flexibility, planning, working memory), was also examined in the context of physiological arousal and injury severity. Lastly, we examined the qualitative aspects of post-concussive symptom reports as a function of head trauma in the university student population because this had not often been examined for persons not undergoing litigation associated with the injury (e.g., Gouvier et al., 2001; Iverson & Lange, 2009).

A quasi-experimental design was employed with two experimental conditions to which participants were randomly assigned: induced-stress/heightened arousal; induced-relaxation/lowered arousal. Using a procedure adapted from Shostak and Peterson (1990) and the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) for the stress condition, the participants were asked to perform a verbal mathematical task under speeded/timed conditions and were told that their performance would be evaluated by a spectator through the one-way mirror. In the relaxation condition, relaxing breathing

techniques were accompanied by guided imagery, restful sounds, aromatherapy, and dimmed lighting. Physiological (i.e., EDA, heart rate, and respiration) and self-reported indices of arousal were recorded (Polygraph Professional Suite, 2008) intermittently throughout a 2.5 hour testing session that was interspersed with blocks of neuropsychological testing.

In line with emotional underarousal, we found that students who reported a history of MHI (N = 51; 56%) produced significantly less resting/baseline EDA than their no-MHI cohort, reported being significantly less aroused/stressed than did students with no-MHI (N = 40; 44%), yet reported significantly more life stressors such as financial or relationship difficulties (Baker & Good, 2010; 2014). In general, students with self-reported MHI produced significantly attenuated physiological arousal (i.e., slower EDA and lower heart rate) and reported less arousal/stress to the experimental manipulations of arousal state (i.e., less heightened arousal [stress] or low arousal [relaxation]) relative to their no-MHI counterparts). We also explored these data as a function of severity of head injury via three groups: no-MHI, MHI-with-ASC, and MHI-with-LOC. This analysis revealed an overall gradient of physiological and self-reported underarousal as a function of severity of injury (i.e., more substantial injury, even lower arousal) at baseline and in response to the arousal manipulations despite reporting more life stressors than their no-MHI cohort (Baker & Good, 2010; 2014).

Notably, students with self-reported MHI did not maintain changes in arousal state as a function of the manipulated arousal for a sufficient period of time which may have affected the sensitivity of detecting effects of manipulated arousal on cognitive measures (i.e., subtests of the Wechsler Memory Scale-III [WMS-III, Wechsler, 1997], the

Wechsler Adult Intelligence Scale – III [Weschler, 1997], the Delis Kaplan Executive Function System [DKEFS; Delis, Kaplin, & Kramer, 2001], the Comprehensive Test of Nonverbal Intelligence [CTONI; Hammill, Pearson, & Wiederholt, 1996]). Nonetheless, students with MHI tended to perform better on a cognitive flexibility task (i.e., Mental Control, WMS-III, 1997) in the stress condition than did students in the relaxation condition, whereas students with no-MHI performed better on this task in the relaxation condition than in the stress condition. Although students with MHI demonstrated a tendency to perform more poorly on cognitive tasks (i.e., working memory, attentional tasks) at baseline before any arousal manipulation, enhanced performance post-arousal manipulation (i.e., psychosocial stress condition) did not reach statistical convention for significance. Lastly, head injury symptoms such as headaches, irritability, and judgment difficulties were assessed via the Post-Concussion Syndrome Checklist (PCSC; Gouvier et al., 1992). Students who acknowledged a history of MHI reported experiencing postconcussive symptoms significantly more often, for longer periods of time, and with greater intensity than students who did not report a MHI. Overall, our findings demonstrate that despite increased reports of experiential life stressors persons with mild head trauma were underaroused in terms of their affective and physiological responses. These findings are suggestive of lasting effects of neural disruption after mild head injury and mirror that found with persons with moderate to severe VMPFC disruption (see Baker & Good, 2010; 2014).

We suggested that this profile of emotional underarousal evidenced for persons with even mild head trauma may be explained by an extension of the somatic marker hypothesis put forward by Damasio and colleagues (1990; 1998). Damasio and

colleagues' somatic marker hypothesis basically emphasizes the importance of somatosensory involvement in successful decision making in which 'gut feelings' guide appropriate decision making. Their research has consistently demonstrated that persons with damage to the VMPFC demonstrate attenuated physiological arousal and poor decision making (e.g., Bechara, Damasio, Damasio, & Anderson, 1994; Bechara et al., 2000). Extending Damasio's theory, persons with MHI may experience a reduction in arousal and affective status as a result of lessened feedback involving 'emotional' somatic markers to or from the OFC/VMPFC. Thus, deficient or disrupted feedback of 'emotional' markers may contribute to the altered perception (e.g., Bechara et al., 2000; Hornak et al., 1996) of stressful experiences and the lowered arousal status. Furthermore, damage to the VMPFC can result in altered communication with the amygdala which typically initiates the stress response (see Kringelbach & Rolls, 2004; Wallis, 2007 for reviews; Barbas et al., 2003) and may result in dysregulated/attenuated stress responsivity. Moreover, it is also possible that this altered perception of typically emotionally-arousing events is related to a more positive, albeit less realistic, self-view (*'the rose-coloured glasses bias'*—Beer et al., 2003; 2006). The studies that comprise this dissertation are an extension of this research. Notably, a major gap in the literature remains regarding physiological and neuroendrocrine indices of emotional functioning (e.g., EDA, salivary cortisol response) as well as self-reported indices of socioemotional functioning (such as emotional intelligence) or other behavioural indices (i.e., affect recognition) following TBI, especially for persons with mild head trauma.

Format and Goals of the Dissertation

Three studies form the basis of this dissertation. Firstly, portions of data from a collaborative research project (i.e., Baker, Dzyundzyak, & Good, 2014) entitled *Individual Differences in Psychosocial and Emotional Functioning in University Students with and without Mild Head Injury* (*N* = 230) form the first study *Emotional and Social Functioning of University Students with and without Mild Head Injury*. The first study provided an exploration of indices of emotional functioning particularly emotional intelligence as well as postconcussive symptom reports in the university population for students with and without self-reported MHI. We also examined whether indices of emotional functioning (i.e., emotional intelligence) would be predictive of self-reported social functioning. In the second study, *Emotional Functioning and Reactivity of University Students as a function of a History of Mild Head Injury* (*N* = 85) we further examined the constructs of emotional intelligence and responsiveness (self-reported and physiological indices) to emotionally-evocative stimuli as a function of a history of mild head trauma. In the third study, *Neuroendocrine and Autonomic Indices of Stress Responsivity for Persons with Traumatic Brain Injury and Mild Head Injury* (*N* = 81), we continued to investigate the underarousal hypothesis, indices of emotional functioning (self-reported and physiological indices), and neurocognitive status (e.g., neuropsychological test performance) of persons with mild and moderate TBI relative to persons with no history of head trauma. In particular, this last study explored measures of salivary cortisol, and stress responsivity of persons with mild and moderate/severe head trauma.

Across this trajectory of research, physiological indices of emotional arousal (e.g., salivary cortisol, EDA, heart rate) as well as self-reported experience of emotional events/functioning have been of primary interest and were explored as a function of severity of injury (e.g., mild or moderate TBI) to examine a continuum of neural disruption (e.g., Alexander, 1995; Iverson & Lange, 2009) and a potential gradient of emotional responsivity. Sex differences as a function of a history of head trauma and stress responsivity were also examined. The models of emotional volatility/lability or attenuated emotional responsiveness following TBI were explored in terms of emotional arousal (self-report and physiological) and overall emotional functioning (e.g., emotional intelligence; affect recognition). Symptom reports often associated with head trauma (e.g., headache, memory difficulties – e.g., Gouvier et al., 1992) were also examined. The goal of this dissertation is to contribute to the state of knowledge of the profile of persons who have sustained a history of head trauma relative to persons without such injury. This research is unique in that socioemotional functioning in terms of indices of stress responses (self-report or physiological) and emotional intelligence is rarely examined in the TBI population; furthermore, to my knowledge this trajectory of research is unique with respect to the emphasis on the continuum of the severity of head injury (also see Alexander, 1995; Iverson & Lange, 2009).

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STUDY 1: EMOTIONAL AND SOCIAL FUNCTIONING OF UNIVERSITY STUDENTS WITH AND WITHOUT MILD HEAD INJURY

Introduction[1]

Mild Head Injury

Adolescents and young adults commonly incur head trauma (see Canadian Institute for Health Information, 2007; Cassidy et al., 2004; Kraus & Chu, 2005; Kraus & Nourjah, 1988; McKinlay et al., 2008; Ryan, O'Jile, Gouvier, Parks-Levy, & Betz, 1996). In the United States, an estimated 475,000 cases of traumatic brain injuries (TBI) occur every year for children and youth 0-14 years of age (Langlois, Rutland-Brown, & Thomas, 2006). Mild TBI is the most common form of these injuries and has been estimated to account for approximately 70 to 90% of all TBIs (Feigin et al., 2013; Iverson & Lange, 2009; Kraus & Nourjah, 1988; Kraus & Chu, 2005; Yeates, 2010). Mild TBI is especially prevalent for children and youth (Yeates, 2010). Mild TBI has been defined by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (ACRM; Kay et al., 1993) as a "traumatically induced physiological disruption of brain function" manifested by a least one of the following criteria of which "at least one must be present: (1) any period of loss of consciousness, (2) any loss of memory for events before or after the event, (3) any alteration in mental state at the time of the injury (e.g., feeling dazed, disoriented, or confused), (4) focal neurological deficits that may or may not be transient" (pp. 86-87). The severity of the injury is characterized by "(1) a loss of consciousness exceeding 30 minutes, (2) a Glascow Coma Scale (GCS) score below 13, and (3) PTA persisting longer than 24 hours" (p. 86-87). However, most persons with 'mild' injuries

[1] Portions of this study have been presented at the 9th and 10th International Brain Injury Association Conference in Edinburgh, Scotland (2012) and San Franciso, California (2014).

do not seek medical treatment (e.g., Segalowitz & Lawson, 1995; Sosin, Sniezek, & Thurman, 1996). If they do, they are typically not admitted to the emergency department and may receive a variable degree of medical treatment (e.g., Mellick, Gerhart, & Whiteneck, 2003). As such, the majority of estimates of mild TBI only include cases of head trauma that have resulted in hospital admissions and do not include self-reports (e.g., Sosin et al., 1996; Teasell, Aubut, Bayley, & Cullen, 2013). Therefore, these incidence rates may be underestimated (Teasell et al., 2013).

For young adults attending university, the self-reported prevalence of mild head trauma (e.g., concussion) ranges from approximately 30 to 56% of students (Baker & Good, 2014; Segalowitz & Lawson, 1995; van Noordt & Good, 2011). Despite the heightened prevalence of mild head injury (MHI) in the university population, limited research has examined the self-reported emotional functioning, physical health (i.e., postconcussive symptom reports), and social functioning of university students with and without self-reported mild head trauma (e.g., Baker & Good, 2014; Gouvier, Cubic, Jones, Brantley, & Cutlip, 1992; Segalowitz & Lawson, 1995; Wang, Chan, & Deng, 2006). The current research focused on this gap in the literature. In this study we examined the profile of postconcussive (PC) symptom reports as a function of a history of head trauma in the university student population. Indices of emotional functioning, primarily emotional intelligence and experience of life stressors, were examined as it is possible that they may be implicated in the sequelae associated with mild head injury in general. Further we examined relationships between PC symptom reports, emotional intelligence and social functioning.

Postconcussive Symptoms

PC symptoms are commonly experienced after mild head trauma (e.g., Bohnen, Jolles, & Twijnstra, 1992; Dikmen, McLean, & Temkin, 1986; Ruff, Camenzuli, & Mueller, 1996) and include affective (i.e., changes in mood), somatic (e.g., headaches, dizziness, blurred vision, hypersensitivity to noise or light), and cognitive symptoms (e.g., memory difficulties, judgment difficulties, attentional difficulties). Collectively, this constellation of symptoms is referred to as postconcussion syndrome (PCS; Binder, 1986; Gouvier et al., 1992; International Classification of Diseases [ICD]-10; World Health Organization, 1992), or depending on the classification system may be termed persistent PCS (Alexander, 1995; ICD-10, World Health Organization, 1992) or postconcussional disorder (PCD; Diagnostic and Statistical Manual of Mental Disorders Fourth Edition-Text Revised [DSM-IV-TR], American Psychiatric Association, 1994). Although the literature suggests (e.g., Binder, Rohling, & Larrabee, 1997; Dikmen, McLean, & Temkin, 1986; Levin et al., 1987; Iverson & Lange, 2009) that the majority of persons with mild TBI make a favourable recovery, a substantial minority of persons (10-20% - Center for Disease Control and Prevention, 2007), referred to as the "miserable minority" (Ruff et al., 1996), continue to experience postconcussive symptoms. For instance, some persons experience postconcussive symptoms up to 1 year (Middleboe et al., 1992), 3 to 5 years (Masson et al., 1996), and even up to 23 years postinjury (Klonoff et al., 1993).

However, PCS/PCD is one of the most controversial disorders and its etiology is often hotly debated in the literature (e.g., Bohnen et al., 1992; Carr, 2007; Frencham, Fox & Mayberry, 2005; Iverson, 2005; King, 2003; Mittenberg & Strauman, 2000; Pertab,

James, & Bigler, 2009; Rohling et al., 2011; Rohling, Larrabee, & Millis, 2012). The issues have focused on concerns regarding the clinical utility of the PCS diagnosis, the specificity, motivation (i.e., litigious), and duration of the symptom reports (e.g., Gouvier, Uddo-Crane, & Brown, 1988; Iverson & Lange, 2003; Lees-Haley & Brown, 1993; Rohling et al., 2011; Wong, Regennitter, Barrios, 1994). Primarily, the nonspecific nature of postconcussive symptoms has been of concern in that the symptoms commonly experienced after mild TBI overlap with other syndromes/disorders (e.g., depression) and are commonly reported in the general population (e.g., Gouvier et al., 1988; Iverson & Lange, 2003; Iverson & McCracken, 1997; Mulhern & McMillian, 2006). As well, the majority of studies on postconcussive symptomology have been conducted with persons receiving clinical treatment for mild TBI (e.g., Ponsford, Wilmott, & Rothwell, 2002) or have included persons involved in ongoing litigation (e.g., Mittenberg, Patton, Canyock, & Condit, 2002) which may introduce motivational components for over or underreporting of symptoms. Furthermore, factors other than neurological disruption, primarily those psychological in nature (i.e., see Mittenberg & Strauman, 2000), have been suggested to account for the development and/or maintenance of long-term experience of postconcussive symptoms (e.g., i.e., longer than 3 months - Clarke, Genat, & Anderson, 2012; cognitive symptoms – Frencham et al., 2005).

In addition, individual difference factors such as sex or experiencing stressful events may play a role in postconcussive symptom reporting, but the findings have been variable. For example, a study by Bazarian, Blyth, Mookerjee, He, and McDermott (2010) found that females had an increased risk of PCS relative to males. Similarly, Sawchyn, Brulot, and Strauss (1999) found that female university students reported

experiencing postconcussive symptoms more often, experienced them with greater intensity, and for longer periods of time than male students whereas other research has not found PC symptom reports to differ significantly between the sexes (e.g., Wang et al., 2003). It has also been reported that, stressful experiences may intensify symptom reporting (e.g., Gouvier et al., 1992). Gouvier and colleagues (1992) found reports of stressors to be significantly related to increased experience of PC symptoms for university students. In addition, being a university student can introduce challenges and research has shown that symptoms of fatigue, irritability, sleep disturbances, and concentration problems are common in a healthy group of university students (e.g., Iverson & Lange, 2003). While some cases of PCS may be related to compensation, gender, student status, or may be exacerbated by stressful experiences, these concerns do not imply that experiences of PC symptoms are not legitimate.

A fundamental issue is whether the profile of persons who have experienced mild head trauma can be distinguished from those of persons who have not experienced head trauma (who may also endorse postconcussive-like symptoms – e.g., see Iverson & Lange, 2003; Sawchyn et al., 1999). Therefore, the current study examined PC symptom reporting in a sample of university students who, to our knowledge, were not complaining about their neurocognitive status, nor were they seeking financial compensation related to their reported mild head trauma and compared their PC report profile to students without a history of head trauma. In a prior study we (see Baker & Good, 2010) demonstrated significant differences in postconcussive symptom reports between university students who had sustained a head injury and those who did not. University students who had sustained a head trauma experienced postconcussive

symptoms more frequently, for longer durations, and with greater intensity than their peers without a history of head trauma. In the current study we expand our examination of reports of postconcussive symptoms in this population to include emotional and social functioning.

Emotional Functioning

Emotional and socioemotional difficulties are often observed following moderate or severe TBI (Bornhofen & McDonald, 2008; Dawson, Levine, Schwartz, & Stuss, 2004; Hawthorne, Gruen, & Kaye, 2009). In particular, difficulties in the recognition and expression of emotions are especially prevalent (e.g., see Spikman, Timmerman, Milders, Veenstra, & Van der Naalt, 2012; Croker & McDonald, 2005; Milders, Fuchs, & Crawford, 2005). Persons with moderate to severe disruption to the brain demonstrate significant challenges in identifying emotional expressions of others (especially those of negative valence) as well as show indices of emotional dysregulation (i.e., flattened affect; Hornak, Rolls, & Wade, 1996; Croker & McDonald, 2005; e.g., emotional outbursts – Hale & Fiorelli, 2004). The research has demonstrated that persons with disruption to the ventromedial prefrontal cortex (VMPFC), a region implicated in the modulation of emotional experience, show reduced emotional experiences both in terms of self-report and autonomic indices (Damasio, Tranel, & Damasio, 1990; 1998). As well, persons with TBI report significantly more difficulty with interpersonal relationships and emotional functioning relative to persons with no history of TBI (Hawthorne et al., 2009; Milders et al., 2003). Although the primary causes of social difficulties postinjury are likely multifocal, it has been proposed that difficulties in social interactions postinjury may be a result of impaired emotional perception and/or aspects of

social cognition (Bornhofen & McDonald, 2008; Ganesalingam, Sanson, Anderson, & Yeates, 2006; Hanten et al., 2008) in domains such as theory of mind and empathy (see Leopold et al., 2011; McLelland & McKinlay, 2013). At the other end of the spectrum of injury severity (Alexander, 1995; Iverson & Lange, 2009) we have examined emotional functioning (e.g., stress responsivity) of university students with and without a history of mild head trauma (Baker & Good, 2014). We have found that students with a self-reported history of MHI (i.e., sufficient to produce an altered state of consciousness – ACRM; Kay et al., 1993) were significantly emotionally underaroused in terms of both physiological (electrodermal activation) and self-report indices despite acknowledging increased experiential life stressors. We suggested that persons with even 'mild' trauma to the head presented in a fashion similar to that of persons with moderate to severe TBI (e.g., Hopkins, Dywan, & Segalowitz, 2002) and/or disruption to the VMPFC (e.g., Damasio et al., 1990) in terms of attenuated indices of emotional functioning relative to persons without head trauma, albeit subtly (Baker & Good, 2010; 2014). We proposed an extension to Damasio and colleagues' (e.g., 1998) "somatic marker hypothesis" that persons with MHI, like persons with moderate/severe TBI, may experience dampened somatic activity that is related to reduced emotional responsivity. In the current study, we continued to test this model of emotional underarousal and its relation to social functioning. To our knowledge, emotional functioning especially in terms of emotional intelligence has yet to be examined with persons with a history of [mild] head trauma (but see Leopold et al., 2001). Nor, to our knowledge, has emotional intelligence been examined in the context of social behaviours with this population.

Emotional intelligence (or 'EI'), in the most broad sense, may be described as emotional regulation which includes the ability to identify one's own emotions as well as recognize or acknowledge others' emotional states (Bar-On, 2000; 2002; Mayer, Salovey, & Caruso, 2000; Salovey & Mayer, 1990). The conceptualization of emotional intelligence has developed from literature focusing on persons with atypical socioemotional development such as that seen in disorders on the Autism Spectrum (e.g., Bar-On, 2000). Similar socioemotional difficulties are often reported in the TBI population (e.g., Dawson et al., 2004; Hawthorne et al., 2009). In particular, damage to the VMPFC has been implicated in socioemotional challenges such as problems with interpersonal relationships and interactions with others (e.g., Eslinger & Damasio, 1985). Moreover, competent emotional intelligence has been suggested to be an essential component of successful social functioning (e.g., Bar-On, 2000; Krueger et al., 2009). To our knowledge very limited research has examined emotional intellect in the TBI population. One study by Leopold et al. (2011) demonstrated that persons with lesions to the VMPFC have shown impaired emotional intelligence and theory of mind abilities. Another study by Krueger et al. (2009) of military veterans with and without penetrating head injuries similarly demonstrated relationships between emotional intelligence competence and prefrontal regions. In general, Krueger et al. found that injury to the prefrontal cortex was related to a diminished perception, understanding, and integration of emotional information as measured by scores on the Mayer Salovey Caruso Emotional Intelligence Test. Krueger et al. as well as others have suggested that emotional challenges following TBI play a role in socioemotional difficulties postinjury (e.g., Bornhofen & McDonald, 2008; Ganesalingam et al., 2006; Hanten et al., 2008; Yeates et

al., 2004). Therefore, in the current study, we explored emotional functioning in terms of emotional intelligence as a function of a history of mild head trauma in a university student population and its potential relationship to social behaviours (as measured by subclinical psychopathic traits). To our knowledge this is the first study to do so. Furthermore, it has been demonstrated that sex differences may exist for emotional intelligence competence (e.g., Naghavi & Redzuan, 2011 – higher for females) or other indices of social functioning (e.g., psychopathic traits – higher for males [Cale & Lilienfeld, 2002; Nicholls, Ogloff, Brink, & Spidel, 2005]). We therefore explored whether a history of head trauma would account for unique variance in social and emotional functioning over and above sex.

Hypotheses

Hypothesis 1. As in Baker and Good (2010), symptoms consistent with postconcussional experiences (as measured on the PCSC; Gouvier et al., 1992) were expected to be experienced with greater frequency, for prolonged periods, and with greater intensity for persons with a history of head injury relative to those with no reported head trauma. We also examined if a history of mild head trauma would account for variance in PC symptom scores over and above sex.

Hypothesis 2. We explored whether measures of stressful life experiences would be related to exacerbated postconcussive symptom reporting (as in Gouvier et al., 1992).

Hypothesis 3. Indices of emotional functioning such as emotion recognition, expression (e.g., Spikman et al., 2012; Bonhofen & McDonald, 2008; Milders et al., 2003), and theory of mind (Leopold et al., 2011) have been shown to be impaired following moderate to severe TBI; therefore, we hypothesized that another index of

emotional functioning – emotional intelligence - would also be attenuated for persons with a history of mild head trauma relative to persons with no reported head trauma. We also expected that a history of head trauma would account for unique variance in emotional intelligence over and above sex.

Hypothesis 4. Because changes in emotional functioning have been suggested to be related to challenges in social functioning for persons with moderate to severe TBI (e.g., Bonhofen & McDonald, 2008), we explored whether indices of emotional intelligence would be predictive of social behaviours for students with mild head trauma. We expected that attenuated EI scores would be related to increased reports of socially undesirable behaviours and that this relationship would be magnified for those with self-reported head trauma.

Methods

Participants

Subjects were 230 university students (males = 39 [17%]; females = 191 [83%]) from Brock University. The mean age of the overall sample was 20.05 years (*SD* = 3.34) and the majority of participants were in the first (*n* = 90 [39%]) or second year (*n* = 51 [22%]) of an undergraduate degree. The study was described as *Individual Differences in Personality and Health* and participants were naïve to the purpose of the study (i.e., see Suhr & Gunstad, 2002; Ozen & Fernandes, 2011).

Materials

The survey package included a multitude of questionnaires (standardized clinical measures; research-based measures – described below) to collect data on self-reported indices of health (including history of head trauma), personality characteristics,

emotional intelligence, life stressors, and PC symptoms. Other measures described elsewhere (e.g., see Baker, Dzyundzyak, & Good, 2014) were also administered but do not form part this dissertation. Notably, with the exception of the demographic questionnaire, the names of the questionnaires were exempt from administration – e.g., postconcussive symptoms were not identified as being attributed to head trauma (see Ozen & Fernandes, 2011).

Everyday Living Questionnaire ([St.Cyr] Baker & Good, 2008). This demographic questionnaire provided detailed information regarding a history of head trauma including altered state of consciousness (ASC), loss of consciousness (LOC), duration of symptom experience, number of head traumas, and other injury-related information such as etiology of the injury. Other information such as age, sex, educational history, and lifestyle (e.g., exercise history) was also collected.

Emotional Intelligence questionnaire (EI; Barchard, 2001a). This 68-item measure of EI provides an index of skills related to emotional expression (positive or negative), emotional intensity, emotional decision making, emotional responsiveness, and empathy for others. Seven subscales comprise this EI index: "positive expressivity, negative expressivity, attending to emotions, emotion-based decision making, responsive joy, responsive distress, and empathic concern". Items are rated on a 5-point scale of 1 *very inaccurate*, 2 *moderately inaccurate*, 3 *neither inaccurate nor accurate*, 4 *moderately accurate*, to 5 *very accurate*. Some items are reverse coded so that higher scores reflect a higher index of emotional competence. Barchard (2001b) reported internal consistency values for each of the 7 subscales ranging from .59 to .81 for males

and .63 to .83 for females. Similar reliability has been reported elsewhere (e.g., from .60 to .82 in Fayombo, 2012).

Life Stressors Scale (St. Cyr [Baker] & Good, 2007 adapted from Holmes & Rahe, 1967). Our modified version of the *Social Readjustment Rating Scale* has 18 items of major stressful life events such experiencing as a loss of a loved one or financial difficulties. Participants reported any of the stressful life events that had occurred in the past 6 months. The total score of this scale is the sum of the weighted items that reflect the relative impact of the stressor(s).

Postconcussion Syndrome Checklist (PCSC; Gouvier et al., 1992). Ten symptoms commonly associated with postconcussive syndrome (headaches, blurred vision, dizziness, aggravated by noise, anxiety, irritability, and memory, attention, and judgement difficulties) comprise the PCSC. Items are rated on a 5 point Likert scale in terms of frequency (1 *not at all* to 5 *all the time*), intensity (1 *not at all* to 5 *crippling*), and duration (1 *not at all* to 5 constant). Ratings for each symptom are summed to form a total score for each symptom dimension (frequency, intensity, duration) as well as a total score across all dimensions.

Self-report of Psychopathy-III (SRP-III; Palhaus, Hemphill, & Hare, in press). The SRP-III (long version) is a 64-item self-report measure of the four facets associated with psychopathic traits: Erratic Lifestyle, Interpersonal Manipulation, Antisocial (or deviant) Behaviours, and Callous Affect (Palhaus et al., in press). Participants rated how well each statement described him/her on a 5 point Likert scale ranging from 1 *very inaccurate* to 5 *very accurate*. The overall scale alpha has been reported to be .91 (Williams, Nathanson, & Paulhus, 2003). Total scores for each facet provide an index of

interpersonal, lifestyle, antisocial, and affective behaviours. This measure is commonly used with university students (e.g., Seibert, Miller, Few, Zeichner, & Lynam, 2011).

Procedure

Participants were recruited from introductory psychology and upper level courses at Brock University. Eligible participants were invited to complete self-administered questionnaire packages in a single session in a group setting with a researcher. Participants were naïve to the purpose of the study and were informed that the primary goal of the study was to examine individual differences in personality and health in university students. Participants were not informed that one of the main variables of the study was a history of head trauma until the debriefing procedure to avoid diagnosis threat (e.g., Suhr & Gunstad, 2002). Participation in the study session was approximately 1.5 hours and participants were given the opportunity to receive course participation research credit for applicable courses at the local university. No monetary honorarium was provided. Study protocol received ethical clearance from the local university's Research Ethics Board (REB # 08-236) (refer to Appendices A and B).

Data analysis

Chi-square analyses and independent t-tests were performed to examine group differences (i.e., no head injury, mild head injury) for the demographic variables. To test for potential differences in PC symptom reports, emotional intelligence, life stressors, and indices of social behaviours (i.e., SRP-III measure) independent *t*-tests were conducted for total scores and subscales. Regression analyses were conducted to examine if MHI history accounted for variance in PC symptom reports over and above other variables such as sex. Bivariate correlations were performed for each group (noMHI, MHI) to

examine the potential relationship between experience of stress and PC symptom reporting. Furthermore, Analysis of Covariance (ANCOVA) was performed to adjust for experience of life stressors on post concussive symptom reporting. Hierarchical regression analyses were conducted to examine if MHI history accounted for variance in emotional intelligence over and above sex. Lastly, the model of emotional functioning (i.e., EI scores) predicting social behaviours was tested as a function of a history of MHI via regression analyses. Statistical Packages for the Social Sciences (SPSS) Version 18.0 (SPSS Inc., 2008) was used for all analyses.

Results

Demographics

Overall, 42% of students reported a prior head trauma event and of these only half reported a loss of consciousness (LOC) associated with the head trauma. Of those that did report a LOC, the majority indicated the LOC to be less than 5 minutes in duration (refer to Table 1.1)[2]. As to be expected for this age group (Cassidy et al., 2004; CIHI, 2006), the majority of students reported sports-related injuries (54.08%; n = 53) and falls (30.62%; n = 30) as the most frequent causes of head trauma (refer to Figure 1.1). Only half of the students reported seeking medical treatment for their injury. The groups were similar on demographic characteristics such as age, sex, and level of education. Students who reported head trauma were younger than students without a history of head injury (*M*age MHI = 19.34 years *SD* = 1.66; *M*age noMHI = 20.58 years *SD* = 4.10, t (183.23) = 2.84, p = .002. Overall there were more females in this sample than males, although the

[2] *Note*. Although still meeting mild TBI criteria (e.g., Kay et al., 1993) four participants (ID # 116, 162, 205, and 226) reported a LOC for greater than 30 minutes but less than 24 hours which may indicate more substantive injury. Therefore, all analyses were also conducted without these participants but no difference in the pattern of findings was observed.

representation of sex across history of head injury groups was proportional (MHI females = 81.6%, males = 18.4%; noMHI females 84.1%; males = 15. 9%), χ^2 (1) = .24, p = .623. Students with and without a history of head trauma did not differ significantly across the number of years of university education, $\chi^2(5) = 8.44$, p = .134.

Table 1.1. *Indicators of Severity of Injury for Self-reported Head Trauma*

n = 98 (42%)		
Mean age at injury	14.65	(3.90)
Mean years since injury	4.79	(4.28)
	n	Percentage
Concussion	56	57
Received medical treatment	49	50
Stitches	15	15
Overnight stay at medical facility	7	7
Altered State of Consciousness	45	46
Loss of Consciousness	53	54
< 5 minutes	40	75.5
< 30 minutes	9	17
>30 minutes but < 24 hours	4	7.5

Note. Numbers in parentheses are standard deviation.

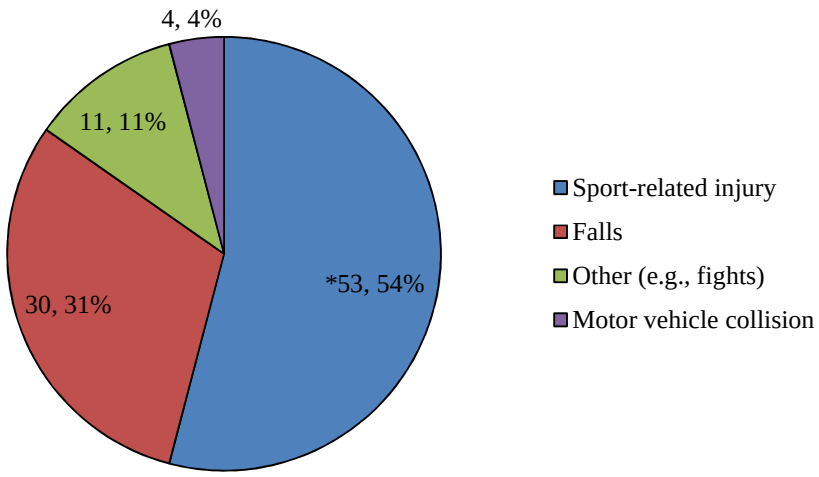

Figure 1.1. Etiology of self-reported head trauma.

**Note.* Frequency is presented followed by percentage.

Postconcussive symptom reports

Consistent with our previous research (Baker and Good, 2010), university students with self-reported mild head trauma had significantly higher total scores on the PCSC, $t(228) = 2.50$, $p = .013$. Students with mild head trauma endorsed postconcussive symptoms significantly more often, $t(228) = 2.36$, $p = .019$, experienced them for longer periods of time, $t(228) = 2.17$, $p = .031$, and with greater intensity, $t(228) = 2.74$, $p = .007$, than students who did not report a history of head trauma (see Figure 1.2 and Table 1.2.). The average scores on the subscales of the PCSC are similar to other studies of postconcussive symptom reports of university students (e.g., Gouvier et al., 1992) and symptom reports were significantly different between head trauma groups (MHI vs. noMHI/controls) in our study (see Table 1.2). Notably, the profile of postconcussive symptom ratings illustrated in Figure 1.3 is such that students with MHI had higher average symptom scores relative to those with no history of head trauma; however, only

experiences of dizziness, $t(228) = 2.12$, $p = .035$, memory problems, $t(228) = 2.52$, $p = .013$, and concentration difficulties, $t(228) = 2.50$, $p = .013$, differed significantly between the groups.

To examine the role of sex in PC symptom reports, a hierarchical multiple regression analysis was conducted (with sex entered as the first predictor) and revealed that a history of head trauma significantly predicted PC symptoms over and above sex, $F(1, 228) = .630$, $p = .013$, but only accounted for 2.68% of the variance. Notably, sex was not a unique predictor in this model. Lastly, a 2 (MHI history: no MHI, MHI) X 2 (Sex: female, male) ANOVA was conducted to examine their potential interaction on PC symptom reporting. The main effect of MHI history remained such that students with prior MHI endorsed significantly more PC experiences than students without prior head injury, $F(1, 226) = 4.57$, $p = .034$. There was no significant main effect of sex, $F(1, 226) = 2.25$, $p = .136$, nor did these factors produce a significant interaction, $F(1, 226) = .10$, $p = .757$.

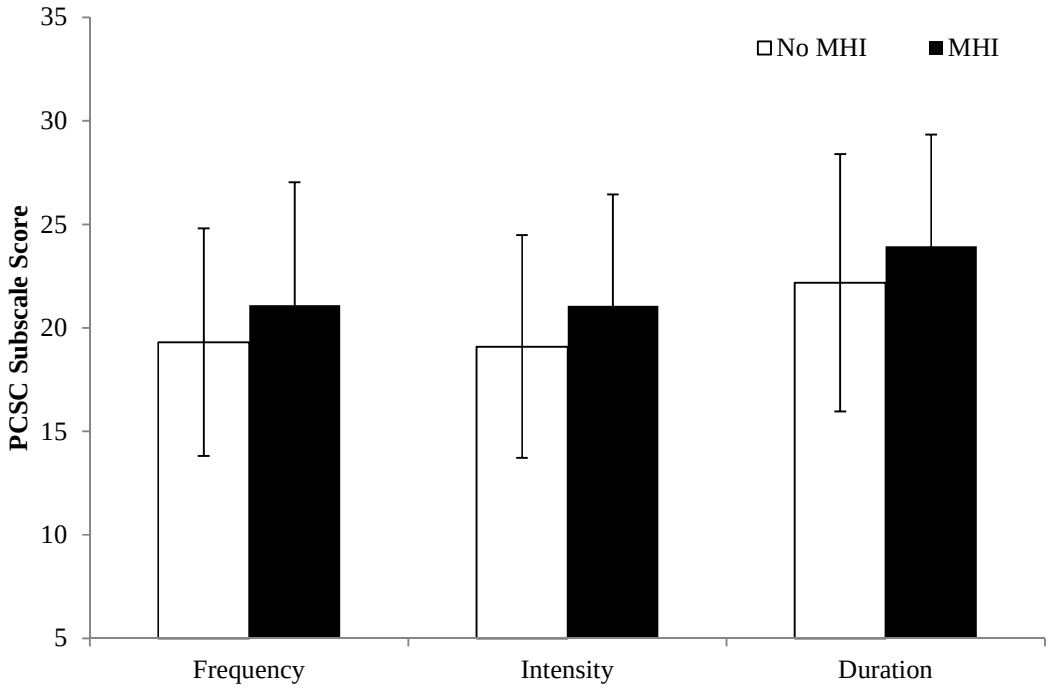

Figure 1.2. Postconcussive symptom reports as a function of a history of self-reported mild head trauma.

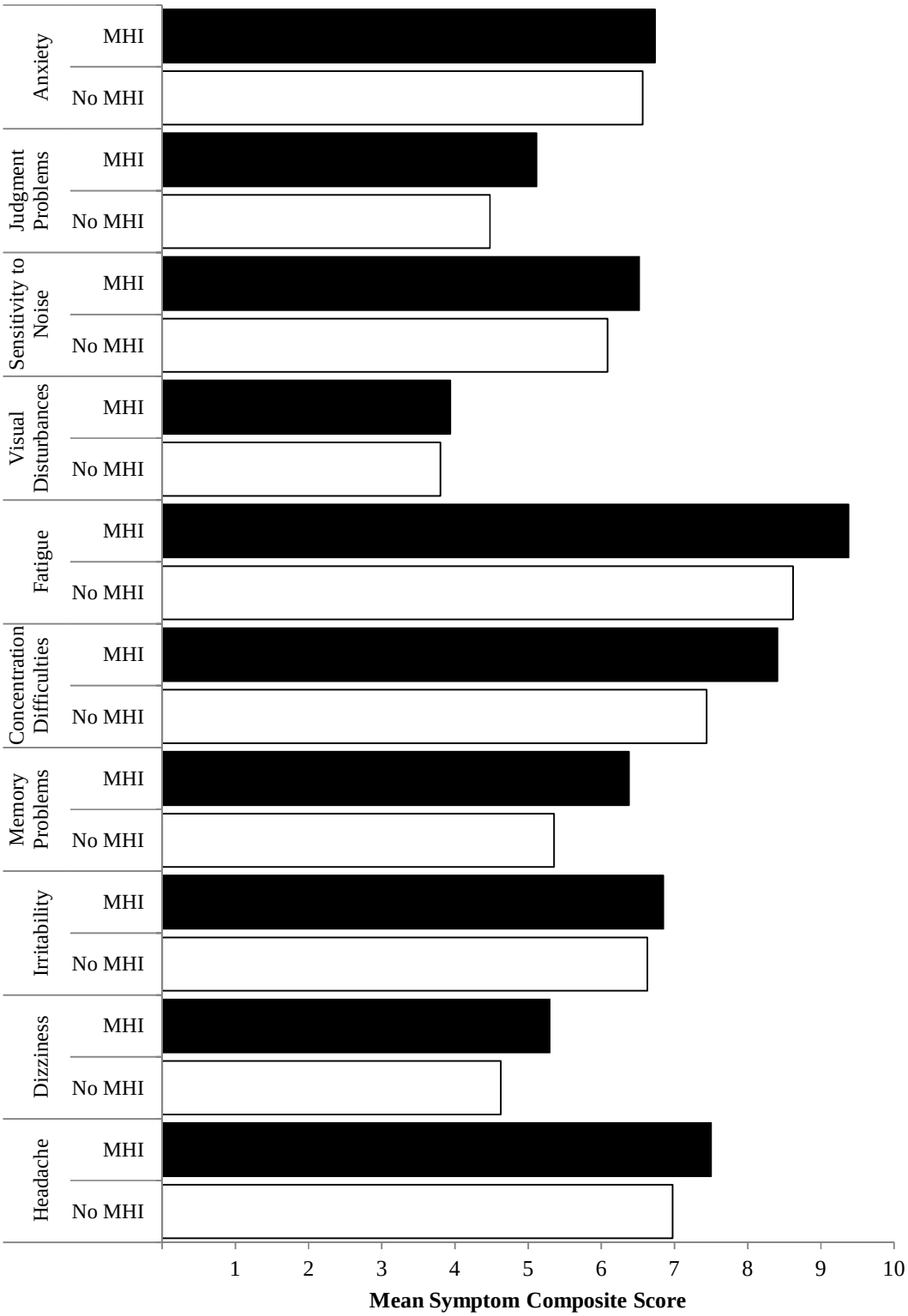

Figure 1.3. Postconcussive symptom report profiles for history of head trauma groups.

Table 1.2. *Comparison of PCSC Scores with Previous Studies Using the PCSC*

	Control Group				Head Trauma Group			
PCSC	Gouvier et al. (1992) *n* = 50	Wong et al. (1994) *n* = 88	Sawchyn et al. (1999) *n* = 247	Current Study *n* = 132	Gouvier et al. (1992) *n* = 47	Wong et al. (1994) *n* = 71 (simulated)	Sawchyn et al. (1999) *n* = 79	Current Study *n* = 98
Total Score	68.45 (13.91)	58.75 (16.36)	67.38 (13.19)	60.58 (16.40)	71.35 (15.98)	72.62 (20.41)	68.15 (16.24)	66.11 (16.70)
Frequency Score	19.76 (4.66)	18.57 (5.26)	20.38 (4.30)	19.32 (5.48)	22.32 (5.28)	23.41 (7.56)	20.52 (5.45)	21.10 (5.94)
Duration Score	24.10 (5.27)	21.20 (6.14)	25.30 (5.37)	22.17 (6.19)	26.06 (5.86)	25.66 (6.98)	26.08 (6.36)	23.94 (6.11)
Intensity Score	21.32 (4.59)	18.98 (5.50)	21.60 (4.52)	19.09 (5.36)	23.30 (5.50)	23.55 (6.60)	21.50 (5.52)	21.06 (5.40)

Life Stressors

University students with a previous MHI reported experiencing significantly more stressful life events than students with no reported head trauma, $t(228) = 2.92$, $p = .004$ (see Figure 1.4), but did not report experiencing more stress than their peers with no head trauma, $t(228) = .47$, $p = .637$.Total scores on the Life Stressors Scale were positively correlated with reports of daily experiences of stress for both students with a prior history of head injury, $r(98) = .20$, $p = .054$, and those with no head injury, $r(132) = .17$, $p = .051$. A 2 (MHI history: no MHI, MHI) X 2 (sex: female, male) ANOVA revealed that there was no significant main effect of sex for reports of stressful events, $F(1, 226) = .37$, $p = .541$, and the main effect of MHI history remained significant, $F(1, 226) = 9.28$, $p = .003$. There was no significant interaction between a history of head injury and sex, $F(1, 226) = 1.56$, $p = .212$, for reports of life stressors.

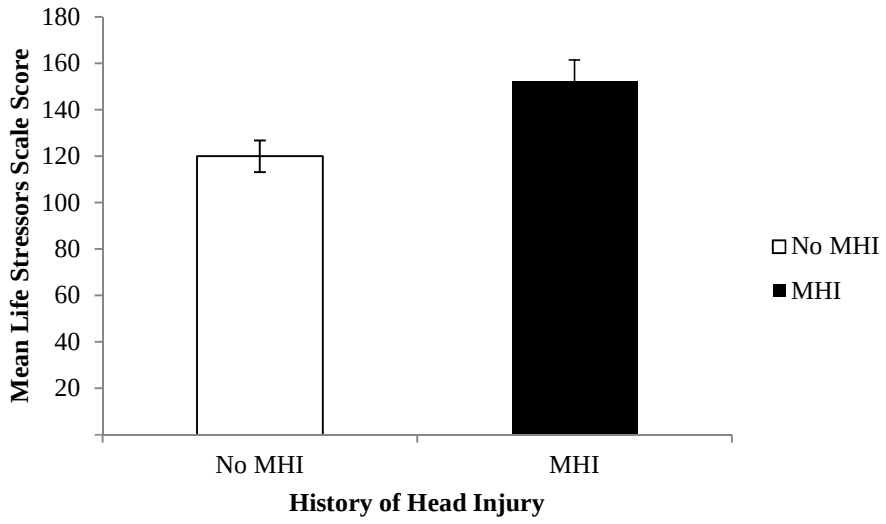

Figure 1.4. Mean Life Stressors Scale Score between history of head trauma groups.

We examined the potential relationship between the experience of stressors and PC symptom reporting (e.g., Gouvier et al., 1992). Bivariate correlations showed an increased experience of life stressors was significantly related to higher PC symptom reports for both students with MHI, $r(98) = .22$, $p = .012$, and without MHI, $r(132) = .22$, $p = .028$. Therefore, we conducted an ANCOVA and found there was a trend for an effect of head trauma history on postconcussive symptom reports after accounting for experience of life stressors, $F(1, 227) = 3.52$, $p = .062$.

Emotional Intelligence

An independent *t*-test showed that the overall score for the EI measure was not significantly different between groups based on a history of mild head trauma, $t(228) = .44$, $p = .661$. EI subscale scores for Positive Expressivity, Negative Expressivity, Attending Emotions, Emotion-Based Decision Making, Responsive Joy, Responsive Distress, and Empathic Concern did not differ significantly as a function of a history of MHI (all *p*s > .05, refer to Table 1.3). Sex differences were evident for the EI total score and subscale scores such that females scored significantly higher than males. A 2-way ANOVA revealed no significant interaction of MHI history by sex on emotional intelligence scores, $F(1, 226) = 2.32$, $p = .129$, but the main effect of sex remained significant, $F(1, 226) = 24.06$, $p < .001$, (MHI history: $F(1, 226) = 2.17$, $p = .143$). Similarly, a hierarchical multiple regression analysis demonstrated that MHI history did not account for a significant proportion of the variance in emotional intelligence total scores over and above sex; overall model: $F(1, 228) = 25.40$, $p < .001$, $R^2 = .10$.

Table 1.3. *Emotional Intelligence Scores between students with and without Mild Head Injury*

	No Head Injury $N = 132$		Mild Head Injury $N = 98$			
Emotional Intelligence	*M*	*SD*	*M*	*SD*	*t*(228)	*p*
Total Scale Score	232.53	26.38	233.08	27.00	-0.44	.661
Subscale						
Positive Expressivity	33.73	4.84	33.61	5.12	0.17	.862
Negative Expressivity	31.84	4.09	31.72	3.49	0.23	.821
Attending Emotions	34.70	5.05	34.48	4.44	0.35	.726
Responsive Joy	38.28	6.47	39.20	6.75	-1.05	.294
Responsive Distress	31.06	6.73	30.84	6.80	0.24	.813
Empathic Concern	35.46	5.96	35.71	6.16	-0.30	.762
Emotion-based Decisions*	27.44	4.84	28.50	3.91	-1.78	.076

**Note*. Equal variances not assumed (corrected degrees of freedom = 226.33).

Social Behaviours

The overall total score for the SRP-III differed significantly between groups such that students with MHI had higher average scores relative to their no head injury peers, $t(228) = 3.21$, $p = .002$. Students with MHI endorsed behaviours consistent with an Erratic Lifestyle, $t\ (228) = 3.25$, $p = .001$, and Antisocial Behaviours, $t(228) = 4.59$, $p < .001$, and differed significantly compared to those with no reported history of head trauma (refer to Figure 1.5). There was a trend observed between groups for scores on the subscale of Callous Affect, $t(228) = 1.79$, $p = .075$, such that students with MHI endorsed characteristics consistent with attenuated emotionality more so than students with no history of head trauma. Interpersonal Manipulation scores did not differ between groups, $t(228) = .68$, $p = .498$. There were significant differences on the SRP-III measure as a function of sex with males scoring higher than females (SRP-III Total Score, $t(228) = 5.07$ $p < .001$; Callous Affect, $t(228) = 4.60$ $p < .001$; Erratic Lifestyle, $t(228) = 4.78$ $p < .001$; Interpersonal Manipulation, $t(228) = 4.44$ $p < .001$; and, Antisocial Behaviour, $t(228) = 2.28$ $p = .024$); however, regression analyses showed that a history of head injury uniquely accounted for 4% of the variance in social functioning (i.e., total SRP-III scores), $F \Delta(2, 227) = 10.35$, $p = .001$, over and above sex (sex uniquely accounted for 10% of the variance) with the overall model accounting for 14.1% of the variance in socially unacceptable behaviours, $F(2, 227) = 18.57$, $p < .001$.

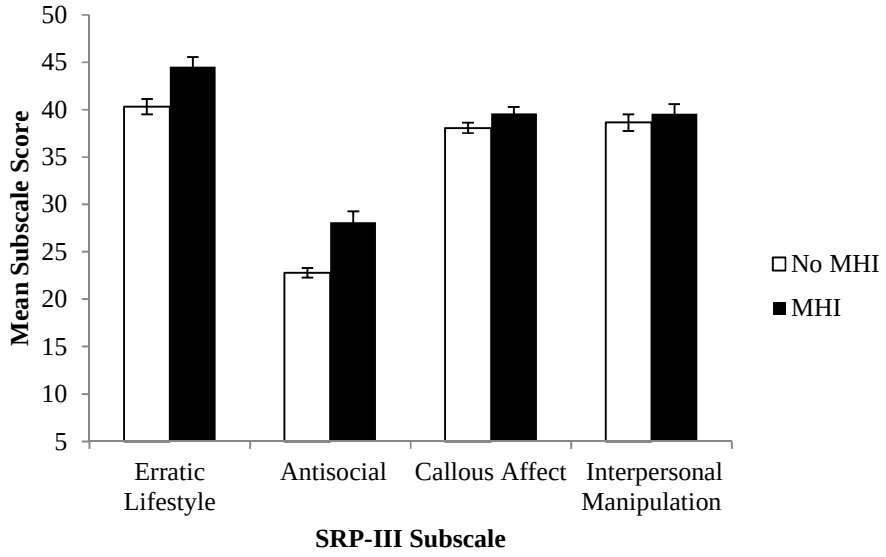

Figure 1.5. Mean SRP-III Subscale Scores between history of head trauma groups.

Emotional Functioning Predicting Social Behaviours

Of particular interest, we examined if an index of emotional functioning (i.e., EI total score) would predict social behaviours (i.e., Antisocial Behaviour subscale of SRP-III) via regression analyses as a function of a history of head trauma. Hierarchical regression analysis demonstrated that total scores on the emotional intelligence measure significantly predicted socially unacceptable behaviours on the first step, $F(1, 228) = 14.84$, $p < .001$, accounting for 5.7% of the variance. The model was also significant when MHI history was included as a predictor on the second step, $F(1, 227) = 20.04$, $p < .001$, which uniquely accounted for 8.9% of the variance in socially unacceptable behaviours. When the interaction of MHI history by total score on the emotional intelligence measure was included on the last step, the model was also significant, $F(1, 226) = 21.52$, $p < .001$ and the interaction term accounted for 7% of the variance (refer to Table 1.4). The overall model including the two predictors and the interaction accounted for 22% of the variance in socially unacceptable behaviours. We conducted separate

regression analyses for each head injury history group. We found that total scores on the emotional intelligence measure significantly predicted socially unacceptable behaviours, but that the model was only significant for students who reported MHI, $F(1, 97) = 23.35$, $p < .001$, accounting for 19.6% of the variance such that lower EI scores were related to poorer social behaviours for students with self-reported mild head trauma, $r(98) = -.44$, $p < .001$, but not for students with no head trauma, $F(1, 130) = .17$, $p = .682$; $r(132) = -.04$, $p = .341$.

Figure1.6a. MHI group

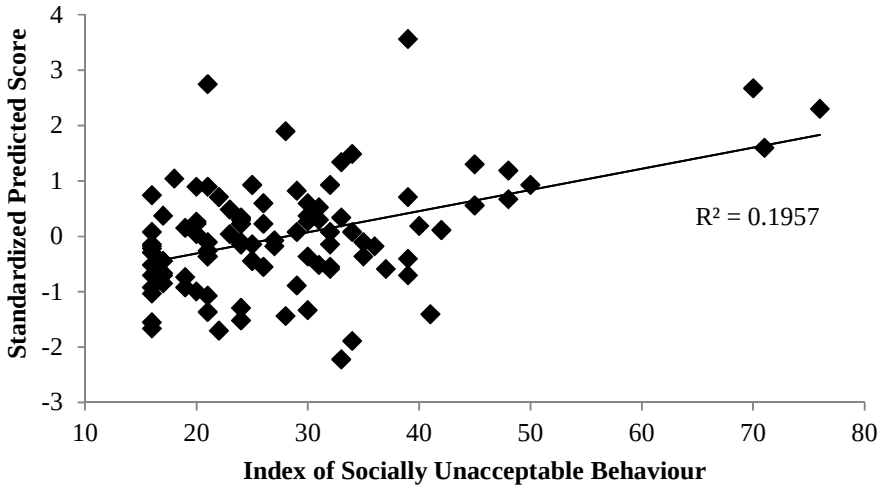

Figure 1.6b. No reported history of MHI group.

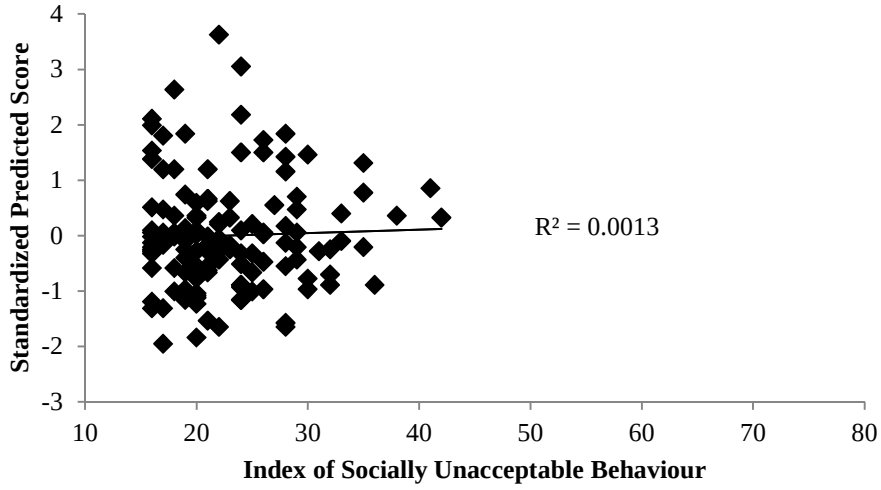

Figure 1.6. Emotional Intelligence Total Scores predicting socially unacceptable behaviours for students with mild head injury.

Table 1.4. ***Hierarchical Multiple Regression Analyses with Emotional Intelligence Total Score and History of Mild Head Injury (MHI) Predicting Scores on the Antisocial Behaviour Subscale of the SRP-III.***

	Model 1			Model 2			Model 3		
Variable	*B*	*SE B*	*β*	*B*	*SE B*	*β*	*B*	*SE B*	*β*
Emotional Intelligence	-.08	.02	-.25	-.09*	.02	-.26*	-.01	-.03	-.02
MHI				5.46	1.12	.30*	47.93	9.49	2.62*
Emotional Intelligence X History of MHI							-.18	.04	-.24*
R^2		.06			.15			.22	
F for change in R^2		14.84*			23.76*			20.28*	

Note. *$p < .05$. **$p < .01$

Discussion

Postconcussive syndrome is a controversial syndrome (see Carr, 2007; Iverson & Lange, 2003). Many factors have been related to increased symptom reporting such as stressful experiences (e.g., Gouvier et al., 1992), sex (female gender is associated with greater symptom reports e.g., Bazarian et al., 2010; Sawchyn et al., 1999), chronic pain (e.g., Iverson & McCracken, 1997), and other variables such as biases or expectations (e.g., Suhr & Gunstad, 2002; Ozen & Fernandes, 2011) or motivation such as ongoing litigation related to the head trauma (e.g., Mittenberg et al., 2002). One of the main goals of the current study was to examine postconcussive symptom reporting in a university student population, the potential relationship of stressful experiences and postconcussive symptom reports, and the possible relationship of sex with symptom reports. Despite the fact that students were naive to the purpose of our study, reports of postconcussive symptom were found to differ between university students who self-reported a history of head trauma and those who did not in the qualitative experience (intensity, duration, and frequency) of the symptoms similar to our previous research (Baker & Good, 2010). Furthermore, the pattern of symptoms was higher for those with a history of MHI across all symptom domains. Although postconcussive symptomology has been suggested to be nonspecific (see Iverson & Lange, 2003) and prevalent in healthy university students (e.g., Iverson & Lange, 2003; Sawchyn et al., 1999), our study demonstrated cognitive symptoms in terms of memory problems and concentration difficulties as well as dizziness significantly differed between students who reported a history of a mild head trauma and those who did not experience a prior head injury event. These symptoms are commonly reported following mild head trauma (e.g., Makdissi, Cantu, Johnston,

McCrory, & Meeuwisse, 2013). Dean, O'Neill, and Sterr (2012) demonstrated a similar pattern of symptom reports with headaches, dizziness, and nausea as the most commonly experienced symptoms (also see Hoffman et al., 2011). Other research (e.g., Wang, Chan & Deng, 2006) has found that university students commonly report PC-like symptoms of fatigue (77%), concentration difficulties (59%), sleep disturbances (50%), and frustration (46%), the qualitative experiences of these symptoms in the current study were found to significantly differ based on a history of a MHI.

Our findings provide corroborating evidence that subjective experiences of PC symptoms persist long after the initial injury and likely are not transient. Overall, students who reported a prior head injury were not in the acute recovery phase because, on average, approximately 5 years had passed since the head trauma event (e.g., similar to Sawchyn et al., 1999), yet those who reported a history of a prior head injury experienced significantly greater, more intense, and prolonged postconcussive symptoms than did students who did not report prior head injury. Similar to these findings, other research also demonstrates that postconcussive symptoms are experienced long after the head injury event. For example, Dikmen, Machamer, Fann, and Temkin (2010) demonstrated that reports of PC symptom experiences were relatively greater for persons who had experienced TBI than a general trauma comparison group at 1 month and even 1 year postinjury (see also Klonoff et al., 1993; Masson et al., 1996; Middleboe et al., 1992). Similarly, another study by Middleboe and colleagues (1992) monitored typical PC symptoms for a year following minor/mild head injury. They found that over half of the persons with mild head trauma in their study reported PC symptom sequelae one year later.

To our knowledge students in our study were not complaining of neurocognitive challenges related to a prior head trauma nor were they involved in litigation related to the injury event. It is important to note that the students were naive to the purpose of the study and were unaware that they were being examined based on a history of a head trauma. We intentionally concealed our interest in a history of head trauma. For example, head injury information was embedded within a variety of health-related questions so it was unlikely that students would associate this information to be a main variable of interest in the study. Moreover, the titles of questionnaires were removed for administration (e.g., Postconcussion Syndrome Questionnaire) so students were unlikely to associate the symptomology with a prior head trauma event. The advertised title of the study also did not indicate our primary interests because other research has found that simply including the term 'mild head injury' can influence reports (Ozen & Fernandes, 2011). Other research has demonstrated that informing students that they are expected to perform poorly on tasks due to a prior head trauma (i.e., "diagnosis threat" – Suhr & Gunstad, 2002) influences their performance (also see Ozen & Fernandes, 2011). Therefore, given the precautionary methods employed in our study, the students likely did not have incentive and/or knowledge to magnify symptom reports (as in Wong et al., 1994 – simulated head injury; Suhr & Gunstad, 2002).

Other researchers have examined concerns regarding the influence of stressful experiences or sex in heightened symptom reporting (Gouvier et al., 1992; Sawchyn et al., 1999). Gouvier et al. (1992) demonstrated that an increased frequency, intensity, and duration of postconcussive symptom reports were related to increased stress for university students. Although we also found that higher reports of postconcussive

symptoms were related to increased experience of life stressors (as in Gouvier et al.), we demonstrated a trend for a history of head trauma accounting for variance in symptom reporting even after controlling for the effect of stressors. Even though increased number of stress events may be related to magnified postconcussive experiences (King, 2003; Gouvier et al., 1992), we suggest that the head trauma in and of itself is the primary underlying factor in postconcussive symptom experience. On another note, female gender has been shown to be related to increased reports of postconcussive symptoms (e.g., Sawchyn et al., 1999), but in our study we did not find sex to be a significant predictor of reports of postconcussive symptoms. A history of head trauma significantly predicted postconcussive symptoms over and above sex. This latter finding is consistent with Wong et al. (1994) and Wang et al. (2006) who also did not find postconcussive-like symptom reports to differ between female and male university students. Again, a history of head trauma appears to play a primary role in postconcussive symptom reporting even when the relationships to factors such as stressful experiences and sex are taken into consideration.

Moreover, it is well accepted that university students report experiencing substantial stress in their day-to-day lives (e.g., Campbell, Svenson, & Jarvis, 1992; Hamaideh, 2009) due to pressures of academic achievement, adjusting to a demanding environment, or other stressors such as relationship difficulties (Hamaideh, 2009; Gouvier et al., 1992) as well as their perceptions of those stressors (e.g., Campbell et al., 1992; Romano, 1992). In our study, although students with self-reported MHI acknowledged significantly more experiential stressors than students without head injury, they reported similar levels of day-to-day stress as their no MHI peers. This latter finding

suggests possibly a decreased perception/impact of emotional life events and perhaps their circumstances for persons with mild head trauma (possibly similar to 'la belle indifference' (Pierre Janet [1849-1947]; see Iezzi, Duckworth, & Adams, 2004; Martelli, Nicholson, & Zasler, 2007). It is possible that students with MHI have an altered and/or more positive perception of their situation (e.g., as in anosagnosia) which is similar to that observed for persons with damage to the orbitofrontal cortex (e.g., Beer, Heerey, Keltner, Scabini, & Knight, 2003). In summary, our findings demonstrate that in a naive sample the postconcussive symptoms following mild head trauma may not be nonspecific, transient, or accounted for by stressful experience or female gender.

A few limitations must be acknowledged. A limitation of the current study is that we did not have objective measures of cognitive abilities (i.e., memory, concentration) nor physical assessment (e.g., of dizziness) to complement the PC symptom reports. Furthermore, we did not examine preinjury characteristics (e.g., personality) and their potential relation to head injury or postconcussive symptom reports. As well, we did not obtain medical records to corroborate self-reported head injury event characteristics. We acknowledge the retrospective nature of the information may pose potential issues regarding the accuracy/details of the information provided and may be biased (e.g., see Harrison, McLaughlin, & Coalter, 1996). However, we used a self-report method because we wanted to capture both persons who did and did not seek medical treatment for their head trauma event (e.g., see Sosin et al., 1996; Teasell et al., 2013). The information provided by students in our study is similar to information that would be obtained during a clinical interview (Kazdin, 2003).

Another primary goal of the current study was to further examine the underarousal hypothesis for students with mild head trauma. In prior research we have demonstrated that persons with MHI presented with attenuated emotional functioning relative to their peers with no history of head injury (see Baker & Good, 2014). In the current study we examined if persons with self-reported MHI would demonstrate differential emotional arousal on another index of emotional functioning i.e., emotional intelligence. Research of persons with moderate to severe TBI has demonstrated impairments in emotional functioning that may be captured under the term 'social cognition' (Adolphs, 2001; McDonald, 2013). For example, persons with moderate to severe TBI demonstrate impairments in their abilities to recognize emotions (Croker & McDonald, 2005; Bornhofen & McDonald, 2008) or provide empathic responses (de Sousa et al., 2011). To our knowledge, very limited research has examined emotional intelligence abilities of persons with TBI. One study by Krueger et al. (2009) has demonstrated reduced emotional competence for persons with disruption to the VMPFC on a measure of emotional intelligence in terms of their ability to perceive, understand, and manage emotional experiences. Based on these findings (e.g., Baker & Good, 2014; Croker & McDonald, 2005; Bornhofen & McDonald, 2008; de Sousa et al., 2011; Leopold et al., 2011), we hypothesized that in the current study persons with mild head trauma would, similar to persons with moderate to severe neural disruption, demonstrate differences in emotional intelligence (as in Krueger et al., 2009) relative to persons with no history of head injury. This hypothesis was not supported in that students' scores on the EI measure did not significantly differ between those with and without self-reported MHI. Sex differences in EI were evident in the current study and were in concert with prior research

(e.g.,. Naghavi & Redzuan, 2011) in that scores on the EI measure were higher for females than males. Differences between persons with and without a history of mild head injury are often relatively subtle (see Panayiotou, Jackson, & Crowe, 2010); therefore, it is possible that potential differences in emotional intelligence in this sample were very slight and/or it is possible that the research-based EI measure was not sensitive. Other, more commonly used, standardized psychometric indices of emotional intellect ability such as the BarOn Emotional Quotient Inventory (Baron, 1997) or the Mayer Salovey Caruso Emotional Intelligence Test (e.g., Mayer, Salovey, Caruso, & Sitarenios, 2003) should be explored in future research with this population because they may provide a more sensitive measurement of this construct.

It has been proposed that difficulties in social functioning may be a result of postinjury difficulties with emotional perception and/or aspects of social cognition (i.e., see Bornhofen & McDonald, 2008; Ganesalingam et al., 2006; Hanten et al., 2008; Leopold et al., 2011; McLelland & McKinlay, 2013; Spikman et al., 2012; 2013; Yeates et al., 2004) for persons with moderate or severe TBI or focal lesions to the frontal lobes (also see Stuss & Knight, 2013). Based on this emerging literature, we were most interested in exploring the predictive ability of emotional intelligence for socially unacceptable behaviours, especially for students who reported a history of mild head trauma. We found that the mean total score on the EI measure significantly predicted socially unacceptable behaviours from the SRP-III, but this model was only significant for students with self-reported MHI. It has been noted that inappropriate social behaviour may be a function of emotional changes postinjury (e.g., see Riggio, 2011). Therefore, this is an important finding from our study and we are not aware of another study that has

examined this relationship in terms of emotional intelligence. Our findings support the proposal that changes in emotional functioning postinjury is a primary factor related to poor social functioning even for persons with a history of mild head trauma. This latter finding also suggests a continuum of injury severity in that social behaviour of persons with mild head trauma mirrors that of persons with more substantive injury as a function of altered emotional functioning.

Furthermore, we demonstrated that based on the SRP-III scores students with MHI endorsed significantly more behaviours consistent with an erratic lifestyle and socially unacceptable actions, as well as illustrated a trend for less affective sensitivity than students with no history of head trauma. Although these behaviours are consistent with psychopathic traits (Hare, 2003; Patrick, 1994) it is important to note that students did not differ significantly on the hallmark characteristic psychopathy – interpersonal manipulation (Hare & Neumann, 2008) – and likely mirror a presentation of "acquired psychopathy" (e.g., Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Blair & Cipolotti, 2000). Moreover, the dimension of 'callous affect' of psychopathy is characteristic of generally deficient affect and lessened sensitivity to or disregard for others (Hare & Neumann, 2008; also see Book, Quinsey, & Langford, 2007) and is consistent with the 'underarousal' that is implicated in TBI especially following VMPFC disruption (i.e., less emotionally responsive, expressive, etc. Anderson et al., 1999; Blair & Cipolotti, 2000; Damasio et al., 1990; 1998; Hopkins et al., 2002). In line with our previous research (Baker & Good, 2014) in which we demonstrated that students with a history of MHI were significantly emotionally underaroused and less responsive to

stressors, the current study illustrated that students with a history of head trauma reported less affective sensitivity/diminished affective experiences (trend).

As has been discussed elsewhere (Baker & Good, 2014) we have suggested that emotional underarousal of persons with MHI mirrors the profile of persons with moderate to severe disruption to the VMPFC, albeit more subtly. Damasio and colleagues have proposed the somatic marker hypothesis (Damasio et al., 1998) in that persons with TBI have altered/lessened somatic feedback i.e., 'gut feeling' to guide decision making and as a result make poor decisions. We have extended Damasio's proposal to persons with MHI (Baker & Good, 2014). It is possible that other aspects of emotional functioning are altered for persons with mild head trauma as well.

In the current study we demonstrated that emotional intelligence is related to poor social functioning for persons with MHI. This finding suggests that a component of social cognition, emotional intelligence (Adolphs, 2001), may predict successful social interactions even for persons with mild head trauma. In conjunction with this finding, students with self-reported MHI tended to acknowledge affective insensitivity relative to their peers without a history of head injury. It is possible that this blunted emotional intellect and callous affect may present as less sensitive emotionality in social situations which could be related to displays of socially unacceptable behaviour for persons with MHI. We suggest that altered emotional somatic activity likely plays a role in poor social interactions. A well designed, more realistic study comparing aspects of social competence and social cognition (interpretation of other's actions) for adults with acquired brain injury (ABI; i.e., persons with moderate or severe TBI or persons who had experienced a cerebrovascular accident) to controls was conducted by Channon and

Crawford (2010). Participants in their study completed a variety of tasks that were designed to measure their ability to understand and generate appropriate behaviours in social interactions as well as assess their social problem-solving ability. Participants read or viewed short scenarios of social interactions that involved scenarios including sarcastic remarks or awkward social situations. For some of the tasks, participants were asked to provide solutions to problematic social situations. Overall, Channon and Crawford found that persons with ABI had significant challenges in interpreting sarcasm, generated poorer (and fewer) solutions to social problems, and had difficulties detecting which aspects of a social interaction were awkward relative to healthy controls. These findings by Channon and Crawford suggest that measures with more ecological validity (such as the measures they developed and used in this study) show promise for illustrating challenges in social competence and social cognition postinjury. Future research should use paradigms such as the one by Channnon and Crawford in conjunction with physiological measures of emotional arousal to further explore Damasio's proposal across the spectrum of TBI severity to further examine the relationship of emotional and social sequelae (Alexander, 1995; Iverson & Lange, 2009).

Although we do not have neuroimaging evidence to demonstrate anatomical, structural, and/or network disruption for our MHI sample, there is good reason to suspect that many persons in our MHI group may have had disturbances in the systems involved in emotion regulation and social cognition (Adolphs, 2001; Beer, John, Scabini, & Knight, 2006; Wallis, 2007; McAllister, 2008). The prefrontal and temporal lobes are especially vulnerable to injury (McAllister, 2008; 2011) and students with MHI demonstrated changes in self-reported socioemotional functioning postinjury in a fashion

similar to persons with substantive disruption and/or lesions to the VMPFC (Damasio et al., 1990; Leopold et al., 2011). Notably, many other aspects that are present in the TBI population such as executive functioning impairments, attentional difficulties, slowed processing speed, memory deficits and other non-cognitive factors likely influence emotional and social functioning postinjury (e.g., Henry, Philips, Crawford, Ietswaart, & Summers, 2006); however, it is beyond the scope of this study to address these issues.

Despite these limitations, our findings illustrate that emotional functioning should be considered a primary variable for problems that interfere with social reintegration, even for persons with mild head injury. Given the exploratory nature of this study, the results should be replicated with persons with mild and moderate/severe TBI groups to further illustrate a continuum of injury severity in the neurobehavioural sequelae postinjury. Additional aspects of social cognition such as theory of mind (e.g., McDonald, 2013) should be further examined in this population. Further examination of the relationship between emotional and social sequelae may also emphasize the importance of holistic interventions postinjury (e.g., see Mateer, Sira, & O'Connell, 2005). Lastly, postconcussive symptom experiences were found to be significantly different for students with and without a prior MHI and were not better accounted for by sex or stressful experiences in a naive sample. These findings suggest that even mild head trauma may have lingering sequelae years postinjury.

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STUDY 2: EMOTIONAL FUNCTIONING AND REACTIVITY OF UNIVERSITY STUDENTS AS A FUNCTION OF A HISTORY OF MILD HEAD INJURY[3]

Introduction

Emotional Sequelae following Traumatic Brain Injury

The sequelae of traumatic injury to the head/brain is multifaceted (Zasler, Katz, & Zafonte, 2007; Ponsford, 2013). Changes in emotional and social behaviours are considered to be the most debilitating sequelae postinjury (Draper, Ponsford, Schonberger, 2007; McDonald, 2005). A number of studies have documented that moderate to severe traumatic brain injury (TBI) is associated with changes in emotional functioning such as impairments in emotion recognition (e.g., Croker & McDonald, 2005; Green, Turner, & Thompson, 2004; Hopkins, Dywan, & Segalowitz, 2002), the ability to infer mental states of others (e.g., Henry, Philips, Crawford, Ietswaart, & Summers, 2006), and a lessened ability to express emotional states (e.g., Dethier, Blairy, Rosenberg, & McDonald, 2013; Mathiesen, Forester, & Svendsen, 2004).

For example, Ietswaart, Milders, Crawford, Currie and Scott (2008) examined emotion recognition abilities in 30 persons with TBI relative to orthopedic controls both shortly after they sustained the TBI and one year later. Emotion recognition deficits in both visual and auditory modalities were evident soon after the TBI. These impairments persisted for at least for one year postinjury, which demonstrated a lasting effect of TBI on emotional functioning. Similarly, Damasio and colleagues (Damasio, Tranel, & Damsio, 1990) have shown that persons with bilateral VMPFC lesions have impaired emotional responses to social stimuli as measured by electrodermal response relative to persons who had brain injuries that did not involve the VMPFC region. Research such as

[3] Portions of this study have been presented at the 10th Annual North American Brain Injury Society's Conference on Brain Injury, Washington, D.C. (Baker & Good, 2012).

that conducted by Damasio and colleagues has implicated the VMPFC and orbitomedial PFC regions in emotional functioning (Damasio et al., 1990; 1998; see also Wallis, 2007), and social cognition in general (Adolphs, 2001; 2003; Shamay-Tsoory, Tomer, Berger et al., 2005; Stuss, Gallup, & Alexander, 2001; Wood, 2003).

The umbrella term of social cognition encompasses the ability to recognize and understand another's mental state as well as to understand other people's intentions and their beliefs – all of which are necessary components of successful social interactions (e.g., Adophs, 2001; 2003; McDonald, 2013; Wood, 2003). These abilities, among others, may be captured in the constructs of emotional intelligence (e.g., Baron, 2000) and empathy (e.g., see Spreng, McKinnon, Mar, & Levine, 2009). The most common reports from family members regarding socioemotional challenges following TBI fall within the domain of impaired social cognition in terms of unemotionality, a lack of concern for others/disinterest, and a lack of empathy (e.g., Milders, Ietswaart, Crawford, & Currie, 2006). Additional literature has corroborated these reports in that persons with TBI have demonstrated impaired empathic abilities (e.g., de Sousa et al., 2010; de Sousa, McDonald, Rushby, Li, Dimoska, & James, 2011; Grattan & Eslinger, 1989; Wells, Dywan, & Dumas, 2005) and theory of mind (e.g., see Spikman, Timmerman, Milders, Veenstra, & van der Naalt, 2012). However, limited research has been conducted to examine the construct of emotional intelligence for persons with TBI (Krueger et al., 2009; Leopold et al., 2011; Baker & Good, 2014a [Study 1]) and, in general, these studies have shown reduced EI relative to persons without head trauma and/or relationships with challenges in social functioning.

Similar to the challenges observed in components of social cognition postinjury (Baker & Good, 2014a; de Sousa et al., 2010; Krueger et al., 2009; Leopold et al., 2011; Milders et al., 2006; Spikman et al., 2012), persons with disruption to the VMPFC regions demonstrate physiological indices of muted emotional responding. For example, lesion studies (e.g., Bechara, Damasio, & Damasio, 2000; Damasio et al., 1990; Tranel & Damasio, 1994) and studies of persons with moderate-to-severe closed head injury have shown reduced electrodermal responsivity to emotional and/or social stimuli (e.g., Hopkins et al., 2002; Hornak, Rolls, & Wade, 1996). It is plausible that the less skilled recognition of and/or response to emotional stimuli for persons with TBI is a function of attenuated somatic feedback (e.g., see Damasio et al., 1998). For example, Hopkins et al. (2002) found that persons with closed head injury had reduced physiological activation in terms of attenuated EDA responsivity especially to emotional facial expressions of fear. de Sousa et al. (2011) have also found reduced autonomic arousal (i.e., EDA) to negative facial expressions – especially angry expressions. Similar research by Croker and McDonald (2005) has shown that persons with severe TBI were unable to successfully identify/label basic negatively-valenced emotions of fear, sadness, and disgust (but were normative for surprise, happiness, and anger). These studies collectively demonstrate attenuated affective sensitivity to socially-relevant stimuli that is of negative valence (i.e., facial expressions of fear, anger, sadness, and disgust).

Another study by de Sousa, McDonald, and Rushby (2012) examined emotional disturbances following severe TBI and their self-reported and physiological responsivity to both positive and negative emotionally-evocative stimuli (films). Participants (N = 21 severe TBI; 25 controls) viewed films that consisted of pleasant, unpleasant, or neutral

content. Physiological responsivity in terms of skin conductance and facial muscle activity were measured and self-reported ratings of valence and arousal of the stimuli were provided. de Sousa and colleagues found that persons with TBI were significantly less emotionally reactive (i.e., produced less EDA response) to negative pictures than persons without TBI. Similarly, persons with TBI rated the stimuli as less arousing than persons without TBI (de Sousa et al., 2012). We suggest their findings also substantiate the underarousal hypothesis (e.g., see Baker & Good, 2014) and provide corroborating evidence for impaired affective responsivity to negative stimuli (as in Hopkins et al., 2002 and so forth).

Even though persons with TBI may be less responsive to emotional stimuli relative to their peers without a TBI (e.g., Hopkins et al., 2002), the research has also demonstrated that exposure to an emotional task resulted in increases in arousal state. For example, in a study by McDonald, Hunt, Henry, Dimoska, and Bornhofen (2010), participants with severe TBI (n = 29) and matched controls (n = 32) were exposed to emotionally-evocative films to elicit the feeling of anger. Participants in this study rated their feelings before and after viewing the emotional film. McDonald et al. found that both groups (TBI; controls) reported an increase in anxiety after exposure to the emotional task. This study suggests that introducing an emotional stimulus may modify self-reports of emotional arousal (in terms of anxiety) even for persons with severe TBI who may be potentially underaroused. Modifying arousal state may have implications for emotional, social, and cognitive functioning (see Baker & Good, 2014b; Cahill, Gorski, & Le, 2003; Lupien, Maheu, Tu, Tiocco, & Schramek, 2007).

However, with respect to persons who have sustained mild head trauma, less is known about emotional functioning and emotional reactivity to affective stimuli (see Moore, Terryberry-Spohr, & Hope, 2006). In one study (Baker & Good, 2014b), we examined whether university students with self-reported mild head injury (MHI – i.e., trauma to the head sufficient to produce an altered state of consciousness – Kay et al., 1993 ACRM criteria) would present with an emotional arousal profile consistent with persons with moderate/severe disruption to the VMPFC who demonstrate flattened affect and attenuated emotional physiological activity (Bechara et al., 2000; Tranel & Damasio, 1994; Hopkins et al., 2002). Overall, we found that university students who reported a history of MHI presented with *emotional underarousal* in that they produced significantly attenuated EDA amplitude and lowered self-reported arousal status than did their peers without a history of head injury, despite reporting significantly more experiences with life stressors (Baker & Good, 2014). Furthermore, those with MHI were significantly less emotionally responsive as indexed by EDA amplitude to the experimental manipulations (psychosocial stressor or relaxation experience) relative to their peers with no history of MHI. We suggest that the emotional underarousal and decreased reactivity after TBI may be implicated in MHI in general (see also Jung & Good, 2007; St. Cyr [Baker] & Good, 2007). Lastly, these effects were evident following a gradient of severity of injury (the more complicated injury, the lower the emotional arousal) (Baker & Good, 2014b).

In the current study we further examined the dampened emotional arousal profile (i.e., underarousal) of students with self-reported MHI and their emotional reactivity to emotionally-evocative stimuli relative to their peers with no reported head injury. We

examined responsivity to static displays of affective pictorial content, specifically the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008) as a function of a history of head trauma (MHI, no history of head injury). There is much evidence to suggest that exposure to affective picture stimuli from the IAPS (Lang et al., 2005; 2008) can result in changes in self-reported and physiological indices of emotional response for persons without head injury in terms of EDA, heart rate, and startle reflex (see Bradley & Lang, 2000 for a review; e.g., Codispoti, Bradley, & Lang, 2001; D'Hondt et al., 2010; and others). Autonomic indices of emotional arousal such as EDA serve as a marker of activation of the sympathetic nervous system (Andreassi, 2007; Critchley, 2009), which is particularly responsive to emotional stimuli (see Kreibig, 2010 for review; Mauss & Robinson, 2009). For example, Codispoti and colleagues (2001) exposed persons to negative/unpleasant, positive/pleasant, and neutral pictures from the IAPS and measured a variety of physiological responses (i.e., heart rate, electromyography [EMG], skin conductance, and the startle reflex) in persons with no history of head injury. Codispoti et al. (2001) demonstrated that persons without head trauma elicit increased electrodermal responses for not only unpleasant, but also pleasant emotional pictures. No significant changes were observed for the magnitude of the EDA response when exposed to neutral pictures relative to the pleasant and unpleasant stimuli. Codispoti and colleagues (as well as many others – e.g., see D'Hondt et al., 2010) have demonstrated affective reactivity to these stimuli.

To our knowledge, responses to static displays of affective pictorial content, specifically the International Affective Picture System (IAPS; Lang et al., 2008) have not been widely used for persons with TBI (i.e., limited research but see de Sousa,

McDonald, Rushby, Li, Dimoska, & James, 2010; Saunders, McDonald, & Richardson, 2006 for use with persons with severe TBI), nor have these stimuli been used to explore/induce changes in emotional arousal (both self-report and physiological indices) and compare responses between persons with and without a history of MHI. For example, de Sousa et al. (2010) found reduced autonomic responding to both positive and negative IAPS pictures for persons with severe TBI relative to control participants. In their study, participants with severe TBI also provided less negative ratings of negative stimuli and rated negative stimuli as less arousing than controls. The findings of de Sousa et al. (2010) are consistent with a presentation of emotional underarousal. In another study, Saunders and colleagues (2006) found that persons with severe TBI produced a diminished startle response to negative IAPS stimuli and also rated the negative stimuli as less arousing relative to controls. Arousal is a core dimension of emotional experience (see James-Lange theory as discussed by Cannon, 1927) which appears to be altered following TBI.

Therefore, in the current study, to explore the continuum of injury severity (mild, moderate, severe TBI – Alexander, 1995; Iverson & Lange, 2009), we examined if affective picture stimuli from the IAPS would be effective in inducing physiological changes for persons with and without a history of MHI and whether responses would vary based on the valence of the stimuli (positive/pleasant; negative/unpleasant; or neutral/ambiguous). Based on the literature of persons with moderate/severe TBI (e.g., Hopkins et al., 2002; Croker & McDonald, 2005) and lesions studies (e.g., Damasio et al., 1990), we expected that persons with MHI would demonstrate reduced affective responsivity to emotional stimuli (as in Baker & Good, 2014), especially for those with

negative valence. We also further explored other indices of emotional functioning in terms of social cognition – primarily emotional intelligence via a standardized measure (Emotional Quotient Inventory; Baron, 1997) and a research-based measure (Emotional Intelligence questionnaire, Barchard, 2001) - as well as empathy (Spreng et al., 2009) as a function of a history of head trauma. Furthermore, we explored the possibility that autonomic responsivity (i.e., EDA) may be related to emotional intellect (see Zysberg, 2012) as a function of history of head injury.

Hypotheses

Hypothesis 1. As demonstrated in previous research of persons who presented with underaroused status (e.g., lesion studies - Bechara et al., 2000; Tranel & Damasio, 1994; Damasio et al., 1990; closed head injury – Hopkins et al., 2002; self-reported mild head trauma – Baker & Good, 2014b; Jung & Good, 2007; St. Cyr [Baker] & Good, 2007), we expected there to be a general decreased physiological and self-reported emotional response in people with head injury (i.e., underarousal) compared to those without head injury, despite increased reports of life stressors (e.g., Baker & Good, 2014; Dawson, Levine, Schwartz, & Stuss, 2004).

Hypothesis 2. Due to evidence of changed emotional responding following head trauma (e.g., emotion recognition; Ietswaart et al., 2008; emotional reactivity post-injury - e.g., Hornak et al., 1996; Hopkins et al., 2002), we hypothesized that emotional responding would vary as a function of a history of self-reported head injury (i.e., lower ratings of emotionally-evocative pictures for persons with MHI; reduced EDA – de Sousa et al. 2012; Hopkins et al., 2002 especially for stimuli of negative valence) and explored other indices of reduced overall emotional capacity (e.g., emotional intelligence, empathy - e.g., Leopold et al., 2011; Spikman et al., 2012; de Sousa et al., 2010; 2011; 2012).

Hypothesis 3. The potential relationship between emotional intelligence measures and emotional arousal state and/or responsivity was also explored (i.e., increased EI related to increased physiological responsivity to emotional stimuli – see Zysberg, 2012).

Methods

Participants

Ninety undergraduate university participants (*M*age = 20.55, *SD* = 2.62) from Brock University were recruited to participate in the research ($N = 86$[4]). Recruitment procedures included advertisements posted on the Brock University Psychology Department Research Participation website (i.e., SONA) and around the university campus. Recruitment statements and advertisements informed participants that the general purpose of the study was to investigate personality and emotional functioning in university students. Participants were not initially informed that one of the variables of interest in this study was previous history of head trauma (e.g., concussion)[5]. Inclusion criteria required participants to be fluent in English and have normal or corrected vision.

Measures

Everyday Living Questionnaire. Demographic, lifestyle, and health-related information is provided via the Everyday Living questionnaire (Baker & Good; Brock University Neuropsychology Research Lab, 2008). Information regarding the participant's history of head injury (i.e., "Have you ever sustained an injury to your head with a force sufficient to alter your consciousness (e.g. dizziness, vomiting, seeing stars,

[4] There were problems with 4 participants' data collection sessions (i.e., physiological recording issues; fire alarm during session; one participant withdrew participation due to time constraints).

[5] Some research suggests that informing participants that head injury is a study variable of interest can influence subsequent performance (Suhr & Gunstad, 2002; 2005). This phenomenon is termed 'diagnosis threat' in that individuals have schemas and representations of what their group membership involves and may behave in ways that confirm these representations (i.e., head injuries are often associated with limitations/changes in functional capacity and this may negatively affect how individuals approach and respond to task demands).

or loss of consciousness, or confusion?" [based on ACRM criteria; Kay et al., 1993] "How did the injury occur?" and other injury-related information) was provided. Demographic characteristics such as sex, age, level of education, mental health, exercise history, and sleep habits was collected.

Life Stressors Scale (adapted by St. Cyr [Baker] & Good, 2007 from the *Social Readjustment Rating Scale* of Holmes & Rahe, 1967). This measure contains a list of major stressful life events such experiencing as a loss of a relationship, illness of a close friend, or starting a new job. Participants report if any of the 18 listed major life stressors have occurred in the past 6 months. A total score is derived by summing each item score to reflect the relative amount of impact of the life stressor. Frequency of endorsing stressful life events is also tallied.

Post-Concussive Syndrome Checklist (PCSC; Gouvier et al., 1992). The PCSC provides an index of the self-reported frequency, intensity, and duration of ten symptoms typically associated with persistent concussions (e.g., headaches, concentration difficulties, irritability). Each symptom is rated with respect to frequency (1 *not at all* to 5 *all the time*), intensity (1 *not at all* to 5 *crippling*) and duration (1 *not at all* to 5 *constant*). Total scores are calculated for all symptom reports.

BarOn Emotional Quotient Inventory (BarOn EQ-i; Baron, 1997). The BarOn EQ-i is a 133 item self-report protected psychological questionnaire that provides an index of emotional-social intelligence. Generally, the BarOn EQ-i is used to assess abilities involving: a) emotional expression, b) emotional awareness and perception, c) emotional regulation/management, d) adapting to change in both intra-and interpersonal situations, and e) expression of positive moods and overall motivation. The BarOn EQ-i

has five major subscales (i.e., "*Intrapersonal, Interpersonal, Stress Management, Adaptability, and General Mood"*) and 15 subscales ("*Intrapersonal*: Self-Regard, Emotional Self-Awareness, Assertiveness, Independence, and Self-Actualization; *Interpersonal*: Empathy, Social Responsibility, Interpersonal Relationship; *Stress Management:* Stress Tolerance, and Impulse Control; *Adaptability:* Reality Testing, Flexibility, and Problem Solving; *General Mood:* Optimism and Happiness"). The questionnaire statements (e.g., "I'm in touch with my emotions; I'm aware of the way I feel") are rated on a 5-point scale (1 *very seldom or not true of me* to 5 *very often true of me or true of me*). Higher total and subscale scores reflect higher emotional intelligence.

Symptom Assessment-45 Questionnaire (*SA-45*, Strategic Advantage Inc., 1998). The SA-45 is a 45-item protected self-report measure that assesses overall symptomatology associated with mental health. The SA-45 provides a total of 9 domain scales (e.g., depression, interpersonal sensitivity) that fashion a *global severity index* and a *positive symptom index* (i.e., number of present symptoms). Persons are asked to rate various statements/symptoms (e.g., "soreness of muscles, feeling blue, difficulty making decisions") with respect to "how much that problem has bothered or distressed you during the past 7 days" on a 5-point scale (1 *not at all*, 2 *a little bit,* 3 *moderately,* 4 *quite a bit,* 5 *extremely*). Higher scores reflect higher psychiatric symptomology for that domain (e.g., depression).

Emotional Intelligence (EI; Barchard, 2001). The EI is a 68-item self-report questionnaire that is used to assess emotional expressiveness, emotional responsiveness, awareness of emotions, empathy, and emotion-based decision making. Each item is rated on a 5-point scale (1 *very inaccurate* to 5 *very accurate*) as to how accurately the

statement describes the individual. Seven subscales of this self-report measure reflect factors associated with emotional intelligence: "*Positive Expressivity, Negative Expressivity, Attending to Emotions, Emotion-based Decision Making, Responsive Joy, Responsive Distress*, and *Empathic Concern"*. A total score for EI is derived with higher scores indicating higher emotional intelligence. Similarly, a total score for each subscale is tallied with certain items reverse coded so that higher scores reflect higher emotional intelligence for that factor. Coefficient alpha reliability estimates range from .53 to .89 for each subscale (see Barchard, 2001). Note that Positive Expressivity and Emotion-based Decision Making subscales have 9, instead of 10, items and these subscales were adjusted for equivalence.

Toronto Empathy Questionnaire (TEQ; Spreng et al., 2009). The TEQ is a 16-item self-report questionnaire that provides an index of empathic abilities (i.e., ability to demonstrate appropriate emotional sensitivity of other's emotional states). Participants rate various statements with respect to how frequently they feel or act in the manner described in the statement (0 *never*, 1 *rarely*, 2 *sometimes*, 3 *often*, 4 *always*). A total score is provided by summing the ratings for all items.

Arousal State Measures

Emotional arousal induction material. The International Affective Picture System (IAPS; Lang et al., 2005; 2008) are commonly used standardized, emotionally-evocative stimuli developed by the NIMH Center for Emotion and Attention (CSEA) at the University of Florida. A total of 123 pictures were selected from the IAPS (Lang et al., 2005; 2008) based on high valence and high arousal ratings from previous studies' normative data (Calvo & Avero, 2009; Lang et al., 2008; Libkuman, Otani, Kern, Viger,

& Novak, 2007). Scenes involving erotica were excluded. To our knowledge these stimuli have not been used with persons with mild head injury.

Psychophysiological measures. As described elsewhere (i.e., Baker & Good, 2014b), Polygraph Professional equipment (Limestone Technologies, 2008) including the Datapac USB^{TM} 16-bit Data Acquisition Instrument with Polygraph Professional Suite Software were used to record EDA, heart rate, and respiration. EDA was recorded via silver-silver chloride plated pads placed on the distal phalynx of the index and fourth fingers of the non-dominant hand. Electrodermal responses were measured in terms of amplitude (i.e., the height of the electrodermal response measured in microsiemens [μ*S*]). Heart rate was recorded via a pulse oximeter on the middle finger of the non-dominant hand and was measured in beats per minute (*bpm*). Although heart rate and respiration data were also collected, EDA was the primary measure of interest[6]. All data were carefully screened and inspected manually for artifact prior to analysis.

Verbal self-report of perceived arousal state. As in Baker and Good (2010; 2014), participants provided a self-report of arousal state (1 *very relaxed* to 10 *very stressed*) prior to and after the emotional arousal induction, and at the end of the testing session.

Emotional arousal manipulation. Participants were asked to view and rate 123 emotionally-laden visual stimuli from the IAPS (Lang et al., 2008). The scenes in the pictures involved either neutral/ambiguous, unpleasant, or pleasant stimuli. The pictures selected from the IAPS stimuli (Lang et al., 2005; 2008) were based on high valence and high arousal ratings from previous studies' normative data (Calvo & Avero, 2009; Lang

[6] Respiration was recorded via pneumatic chest bands with the upper band placed at the level of the sternum and the lower band across the abdomen. Respiration was measured in cycles per minute.

et al., 2008; Libkuman et al., 2007). Pictures of persons, animals, and inanimate objects that had high arousal (i.e., excited) and high valence ratings (i.e., positive and negative affect); as well, ambiguous/neutral stimuli were selected (Lang et al., 2005; 2008; Libkuman et al., 2007; Mikels et al., 2005)[7]. The IAPS stimuli (negative, positive, or ambiguous) were randomly presented on a Sony computer screen and the participant rated each picture accordingly via computer response with his/her dominant hand. Participants rated the pictures on 4 Likert scales (arousal and valence scales are derived from the Self-Assessment Manikin (SAM; Bradley & Lang, 1994): arousal (low/calm to high/excited), valence/pleasure (unpleasant/unhappy to pleasant/happy), intensity (not intense to extremely intense), and empathy (no empathy to significant empathy).

Procedures

Study protocol received clearance from the institutional Research Ethics Board (#09-236) (see Appendix C). Participants were tested individually at Brock University (refer to Appendix D). Informed consent was obtained from all participants. All participants were advised that physiological measures would be collected for heart rate, respiration and electrodermal responses (via finger bands around two fingers of the non-dominant hand, pulse oximeter, and respiration bands – Polygraph Professional, 2008). Once the participant was comfortably fitted with the physiological recording equipment, a baseline 3-minute recording was taken and the participant was asked to self-report on his/her arousal state (i.e., how he/she feels on a scale of 1 to 10 [1 *very relaxed* to 10 *very stressed*]). Physiological recordings of arousal state and self-reports of arousal were taken

[7] Note that both positive and negative stimuli were also selected because research has demonstrated that the amygdala responds preferentially to the intensity, rather than the valence of the stimuli (e.g., Garavan, Pendergrass, Ross, Stein, & Risinger, 2001).

intermittently throughout the testing session (e.g., prior to and after emotional arousal induction and the final recording).

Participants then received instructions (see Appendix E) regarding the emotional arousal induction (i.e., viewing pictures of pleasant, unpleasant, or ambiguous scenes; rating the stimuli [as in Lang et al., 2005; 2008]). Participants were informed that we were interested in both physical (i.e., physiological) and self-reported responses to life events such as the ones depicted in the stimuli. Stimuli were displayed individually on a 22" monitor for approximately 5 seconds with a viewing distance of approximately 60 cm. Stimuli dimensions were 25 cm by 25 cm. Participants viewed the pictorial stimuli and rated each picture on 4 Likert scales: arousal, valence/pleasure, intensity, and empathy. Physiological activity recordings were taken throughout the viewing and rating of the pictures. Physiological data were segmented to provide indices of sympathetic nervous system activation during presentation of stimuli, and during response phase (i.e., rating of each picture). The emotional arousal induction lasted approximately 40 minutes with continuous physiological recording throughout with variable sampling.

Following the emotional arousal induction, participants completed self-report questionnaires for approximately 30 minutes to obtain information regarding head injury history, mental health, and socioemotional functioning (BarON EQ-i, Bar-on, 1997; STAI, Spielberger, 1983; Emotional Intelligence, Barchard, 2001; Symptom Assessment-45 Questionnaire, SA-45, Strategic Advantage, 1998; Toronto Empathy Questionnaire, TEQ, Spreng et al., 2009; Post-concussion Syndrome Checklist, PCSC, Gouvier et al., 1992; and, Everyday Living Questionnaire, Brock University Neuropsychology Cognitive Research Lab, 2008). After completion of the self-report measures, a final

physiological activity recording and self-report of arousal state was obtained. Overall, participation in this study (including time for acquisition of informed consent and debriefing procedures) was approximately 1.5 hours. Participants were thanked for their time and participation in the study and were invited to view the results of the study at its completion. Participants were provided with the opportunity to receive research participation credits for applicable courses at the university. No monetary honorarium was provided.

Data analysis

This protocol is a quasi-experimental mixed-model design (between groups variable is history of head injury). Pearson Chi-Square statistic and t-tests were used to compare demographic information between students with and without MHI (e.g., to compare level of education or age). To examine group differences (MHI history, no-MHI history) for continuous measures, t-tests, one-way analysis of variance (ANOVA) and factorial ANOVAs were conducted.

To examine the underarousal hypothesis, baseline arousal (physiological [i.e., EDA] and/or self-reported indices) was examined as a function of MHI history (MHI, no-MHI) via t-tests. Mixed model ANOVAs were conducted for physiological and self-reported measures of arousal to examine differences between head trauma groups and/or responsivity across the testing session. This analysis permits examination of the underarousal hypothesis. We are primarily interested if physiological or self-reported responses to the arousal manipulation varied as a function of MHI history and hypothesized dampened responding for persons with MHI, especially to negative emotional stimuli. Valence, arousal, intensity, and empathy ratings of emotionally-

arousing stimuli were examined in separate 2 x 3 Mixed model ANOVAs as a function of stimulus category (positive valence, negative valence, ambiguous) and MHI history (MHI, no-MHI). The average ratings (valence, arousal, intensity, empathy) of each group (MHI, no-MHI) were similar to prior norms (i.e., Calvo & Avero, 2009; Lang et al., 2008; Libkuman et al., 2007). Response time to the emotionally-arousing stimuli was examined via a 2 x 3 Mixed model ANOVA as a function of stimulus category (positive valence, negative valence, ambiguous) and MHI history (MHI or no-MHI). We also examined whether the manipulation produced significant increases in physiological responses post-induction. Overall emotional functioning as indexed by emotional intelligence measures (i.e., EQ-I; EI), empathic ability (i.e., TEQ), and psychiatric health (i.e., SA-45) was examined for group differences (i.e., lessened emotional responding) via t-tests. Emotional intelligence, particularly the EQ-i, was analyzed for potential influence in emotional arousal state and/or responsivity via regression analysis as a function of a history of head trauma. All physiological data were carefully inspected for artifact. Statistical analyses were conducted with SPSS version 18.0 (SPSS Inc., 2008).

Results

Demographics

Over a third (34.9% *N* = 30) of the university students reported a history of MHI (refer to Table 2.1.) occurring at approximately 16 years old (*SD* = 5.79). More than half of these students reported a loss of consciousness (LOC) associated with the head trauma (63.3% *N* = 19) and the remaining experienced only an altered state of consciousness (ASC; 36.7% *N* = 11). As well, approximately half of the students (46.7% *N* = 14) reported that they experienced symptoms of dizziness, or feeling dazed or confused for

more than 20 minutes following the head trauma event. All the reported LOC periods were under 30 minutes in duration and therefore meet criteria for 'mild' head injury (ACRM; Kay et al., 1993). Of these 78.9% (N = 15) experienced the LOC for less than 5 minutes so they are at the 'milder' end of the continuum of injury severity (Alexander, 1995; Iverson & Lange, 2009). Students most commonly reported falls (N = 13; 43.3%) as the primary cause of head injury, followed by injuries occurring from sporting activities (N = 7; 23.3%), and other causes (e.g., fighting; N = 7; 23.3%) (refer to Figure 2.1). Forty percent (N = 12) reported receiving medical treatment for their head injury and had incurred the injury approximately 5 years earlier. Over a third of students (36.6%; N = 11) reported more than one mild head injury event.

There was equal proportion of males and females with self-reported MHI (females N =15; males = 15); but, there was more female representation in the noMHI group (females N = 41; males = 15), $\chi^2(1) = 4.64$, $p = .031$. The students with reported history of head trauma were older, Mage MHI = 21.40 years SD = 3.09, than students who did not report any head trauma, Mage noMHI = 20.09 years SD = 2.22, $t(45.38) = 2.06$, $p = .046$. Years of education were proportionally represented between MHI history groups, $\chi^2(5) = 4.17$, $p = .525$.

Table 2.1. *Indicators of Severity of Injury for Self-reported Mild Head Injury*

n = 30 (35%)		
Mean age at injury	15.77	(5.79)
Mean years since injury	5.29	(6.04)
	n	Percentage
Concussion	14	46.6
Received medical treatment	12	40.0
Stitches	4	13.3
Overnight stay at medical facility	2	6.7
Altered State of Consciousness	11	36.7
Loss of Consciousness	19	63.3
< 5 minutes	15	78.9
< 30 minutes	4	21.1

Note. Numbers in parentheses are standard deviation.

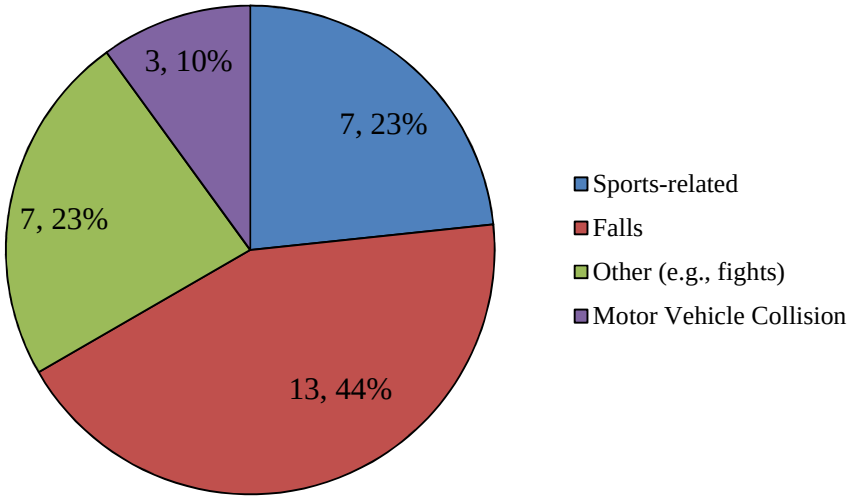

Figure 2.1. Etiology of self-reported head trauma.

Note. Frequency of etiology is displayed followed by percentage.

Physiological Indices of Emotional Arousal

We replicated the pattern of our previous findings (Baker & Good, 2014b) in that students who reported a history of MHI were significantly emotionally underaroused in terms of EDA amplitude, MHI = .89 *uS*, *SD* = .80 *uS*; noMHI = 2.29 *uS*, *SD* = 1.32 *uS*, $t(84) = 5.31$, $p < .001$, and reported the expected pattern, although not significantly so, of experiencing more life stressors than did students with no reported MHI, mean total life stressors score: MHI = 131.33, *SD* = 90.98; noMHI = 102.66, *SD* = 73.15, $t(84) = 1.59$, $p = .11$. Similarly, physiological emotional arousal (i.e., EDA) status differed as a function of severity of injury (i.e., no MHI [*n* = 56], MHI with ASC [*n* = 11]; MHI with LOC [*n* = 19]) in that both head injury groups produced smaller EDA resting responses than did students with no reported head injury, $F(1, 83) = 14.10$, $p < .001$. Multiple comparisons

via Least Significant Difference (LSD) revealed that although students with LOC had lower mean resting EDA than did students with an ASC associated with their injury, these groups were not significantly different from one another, *p*s > .05, but did differ significantly from the students with noMHI who produced larger EDA responses, *p*s < .001.

Self-reported Indices of Emotional Functioning

We examined whether indices of emotional intelligence (a standardized clinical measure [i.e., EQ-i], and research-based measures [i.e., EI]) and empathy (i.e., TEQ) varied for students with and without a history of head injury. Based on the evidence of reduced emotional capacity for persons with head trauma (e.g., Spikman et al., 2012; Leopold et al., 2011) we expected lower scores on these indices of emotional functioning for students with self-reported MHI relative to their peers with no reported head injury. Across both EI measures, we did not find strong support for this hypothesis.

However, in line with our expectations, students with MHI reported decreased responsivity to others' distress relative to their no-head injury peers, $t(84) = 1.77$, $p = .081$ (EI subscale Responsive Distress). Students with MHI had significantly higher positive expressivity relative to those without MHI, $t(84)$ 2.32, $p = .023$. All other EI subscales did not differ between groups as a function of head injury history (see Table 2.2). For the standardized measure of emotional intelligence, the EQ-i, no significant differences were observed as a function of a history of head injury, *p*s > .05. Furthermore, empathic abilities as measured via total score on the TEQ did not differ significantly between MHI history groups, $t(84) = .60$, $p = .55$. Emotional functioning was also examined in terms of an index of psychiatric health via scores on the Global Severity

Index of the SA-45 and all SA-45 subscales (somatization, psychoticism, phobic anxiety, paranoid ideations, obsessive compulsiveness, interpersonal sensitivity, hostility, depression, and anxiety) did not differ significantly between students with and without a history of head trauma (see Table 2.3).

Table 2.2. *Emotional Intelligence Scores between history of head injury groups*

	No Head Injury $N = 56$		Mild Head Injury $N = 30$			
Emotional Intelligence	*M*	*SD*	*M*	*SD*	*t*(84)	*p*
Total Scale Score	231.57	28.40	228.93	22.87	-0.47	.641
Subscale						
Positive Expressivity	31.38	5.49	34.00	3.91	-2.32	.023
Negative Expressivity	31.71	3.42	30.67	4.10	1.26	.210
Attending Emotions	34.27	4.34	35.13	5.71	-0.79	.433
Responsive Joy	37.29	5.49	38.63	6.13	-0.04	.301
Responsive Distress	31.18	5.14	28.87	6.83	1.77	.081
Empathic Concern	35.50	5.54	36.10	6.95	-0.44	.663
Emotion-based Decisions	27.60	4.02	28.17	4.93	-0.57	.572

Table 2.3. *Symptom Assessment Questionnaire -45 scores between history of head injury groups*

	No Head Injury $N = 56$		Mild Head Injury $N = 30$			
	M	*SD*	*M*	*SD*	*t*(84)	*p*
Global Severity Index	81.78	22.28	84.73	23.80	-.57	.570
Subscale						
Anxiety	8.68	2.87	8.90	3.23	-.33	.745
Depression	11.45	4.66	10.90	3.65	.56	.579
Hostility	7.45	2.64	8.23	3.60	-1.16	.251
Interpersonal Sensitivity	10.07	3.85	10.73	4.58	-.71	.479
Obsessive Compulsive	11.87	4.08	12.10	4.10	-.24	.808
Paranoid Ideations	8.98	3.46	10.23	3.26	-1.63	.107
Phobic Anxiety	6.84	2.65	7.53	3.79	.99	.324
Psychoticism	6.98	2.70	6.86	2.61	.19	.849
Somatization	9.46	3.51	9.23	4.14	.27	.786

Responsivity to the Emotional Arousal Induction

Self-reported arousal state in response to the emotional arousal induction. A mixed model 2 X 3 ANOVA (time: pre-arousal manipulation, post-arousal manipulation, end of testing session) with MHI history (noMHI, MHI) as the between-subjects variable revealed that self-reported arousal increased in response to the emotional arousal manipulation across the testing session, $F(2, 168) = 26.09$, $p < .001$. Multiple comparisons demonstrated that students' arousal state differed significantly from pre-to-post manipulation, $p < .001$, and returned to their previous self-reported arousal level after the manipulation, $p = .759$. Although we expected reduced self-reported arousal for the MHI group, there was no main effect of MHI history, $F(1, 84) = .53$, $p = .468$, and there was no significant interaction of time by history of head injury, $F(2, 168) = 1.30$, $p = .274$ (see Figure 2.2).

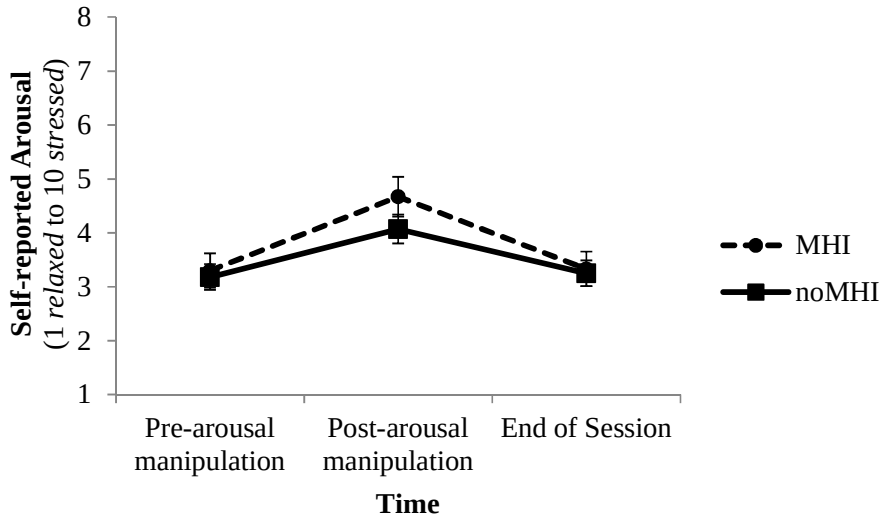

Figure 2.2. Self-reported arousal state across time between MHI and noMHI groups.

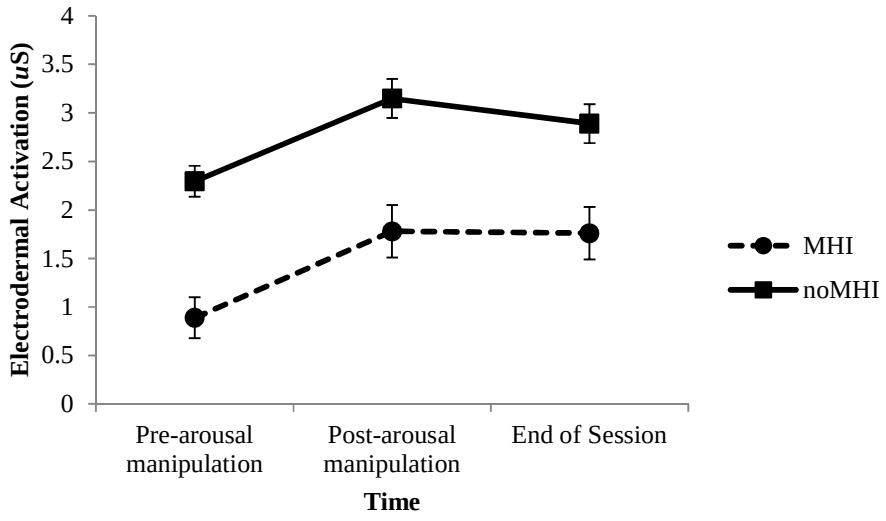

Figure 2.3. EDA amplitude (*u*S) in response to the emotional arousal induction between MHI and noMHI groups.

Physiological arousal state in response to the emotional arousal induction. Notably, and as expected, exposure to the emotionally-evocative stimuli resulted in significant changes in physiological emotional arousal in terms of EDA amplitude, $F(2, 168) = 19.46$, $p < .001$, (refer to Figure 2.3.) such that EDA amplitude increased significantly from pre-to-post manipulation, (multiple comparisons LSD) $p < .001$, and differed significantly from baseline to the end of the testing session, $p < .001$, and this changed arousal status was maintained for approximately 30 minutes (multiple comparison from post-manipulation to the end of the testing session was *ns*). There was no significant interaction between head injury history and EDA amplitude response across time, $F(2, 168) = .51$, $p = .602$; however, a main effect of MHI history was evident such that students with self-reported MHI produced significantly lower EDA amplitude than students with no history of head trauma, $F(1, 84) = 24.94$, $p < .001$.

Ratings, Reaction Time, and Average Responses to the Types of Stimuli

2 (MHI history: noMHI, MHI) X 3 (stimuli type: positive, negative, neutral) mixed model ANOVAs were conducted to examine the possibility of reduced responsivity of students with MHI. The ratings of the emotionally evocative-stimuli (i.e., IAPS) in terms of arousal, valence, intensity, and empathy did not differ significantly for students with and without self-reported MHI, *p*s > .05 (see Figure 2.4), although higher ratings were observed overall for negative, followed by positive, and ambiguous stimuli (see Figure 2.4). These findings did not support our hypothesis. Similarly, the reaction time to each type of image (positive, negative, or ambiguous) did not differ as a function of a history of head injury, *p*s > .05, (see Figure 2.5).

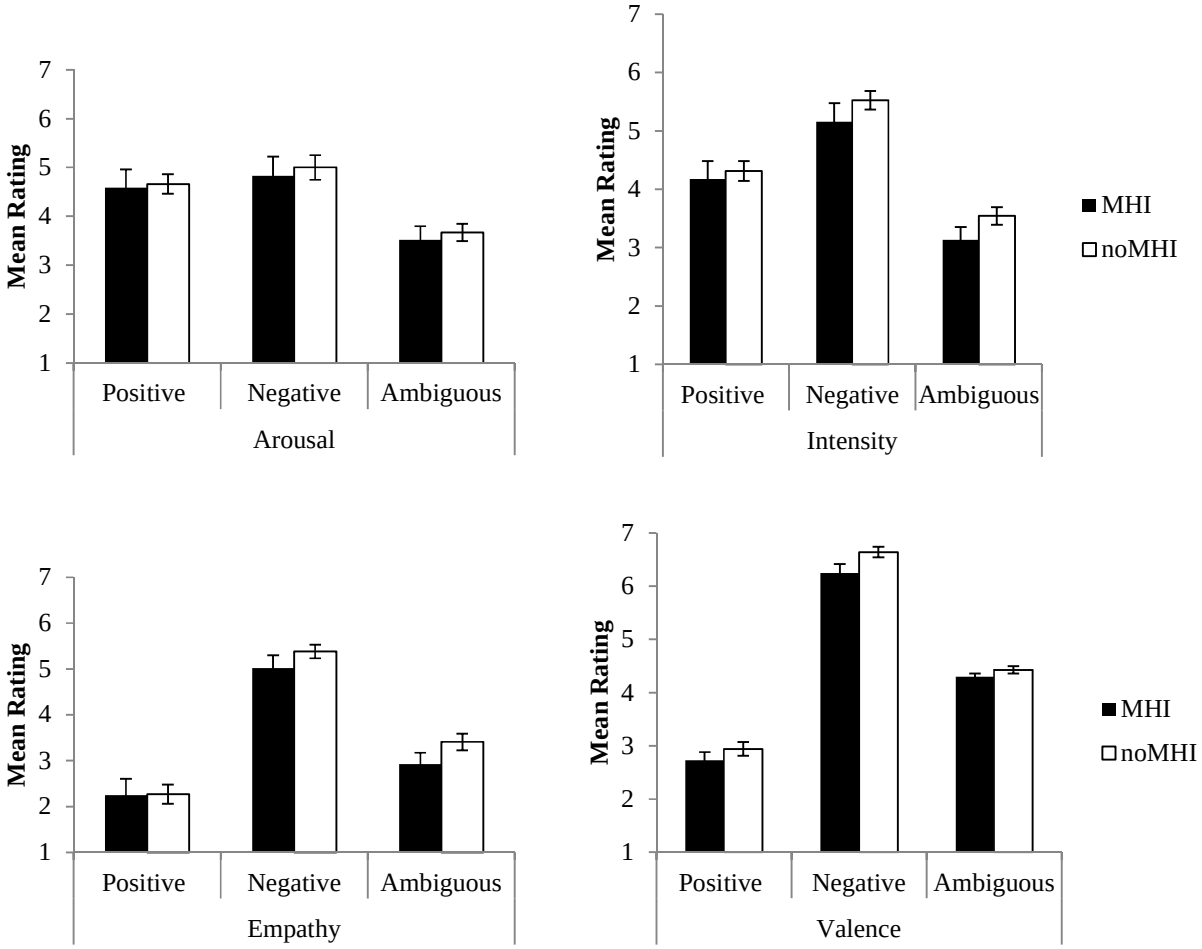

Figure 2.4. Mean ratings (arousal, valence, intensity, empathy) of type of stimulus (positive, negative, ambiguous) between history of head trauma groups.

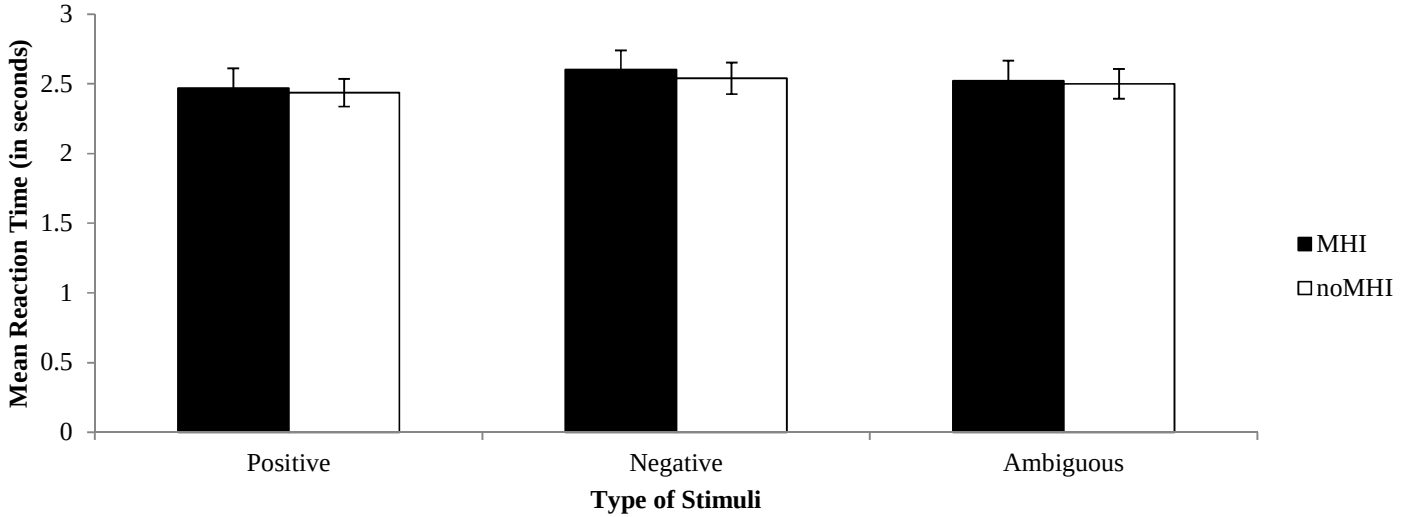

Figure 2.5. Mean reaction time to type of stimulus (positive, negative, ambiguous) between history of head trauma groups.

We hypothesized that, like persons with moderate to severe TBI, students with MHI would be less emotionally responsive to the emotionally-evocative stimuli than would be students with no history of head trauma, especially for stimuli of negative valence. We examined this hypothesis via a 3 X 2 mixed model ANOVA with type of emotional stimuli (positive, negative, or ambiguous) as the repeated measure and MHI history (no MHI, MHI) as the between-subjects variable on EDA amplitude. A significant 2-way interaction of type of stimulus by head injury history was shown for EDA amplitude, $F(2, 168) = 8.87$, $p < .001$. To follow up this significant interaction, repeated measure ANOVAs were conducted separately for each group (no MHI, MHI). We found that for students with self-reported MHI, EDA amplitude did not change significantly as a function of the stimulus type, $F(2, 58) = .14$, $p = .870$, yet students with no history of head trauma produced significantly different EDA responses to the negative, positive, and ambiguous/neutral stimuli, $F(2, 110) = 18.05$, $p < .001$. Multiple comparisons revealed that negative stimuli produced the largest response relative to positive and ambiguous stimuli – all three stimulus types produced significantly varied EDA responses for students without MHI, ps $< .001$. This finding demonstrates flat emotional responsivity in terms of physiological activity for students with self-reported MHI across all stimuli types (refer to Figure 2.6).

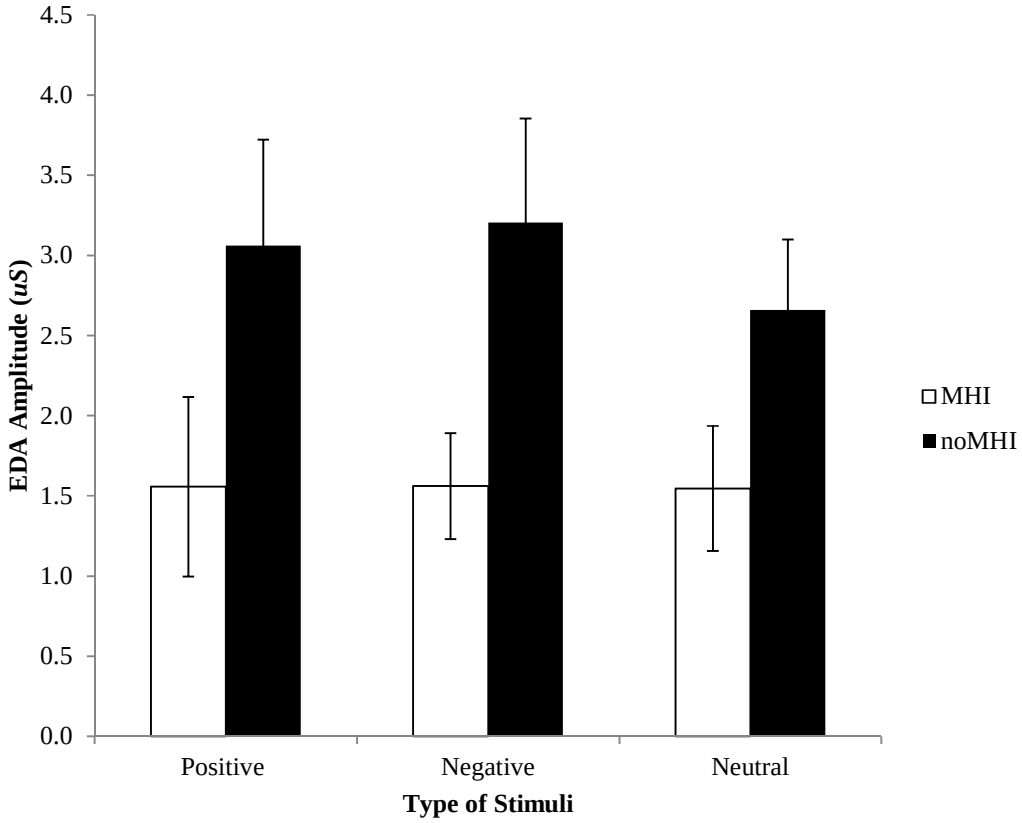

Figure 2.6. EDA amplitude to emotional stimuli only differed for students with no history of mild head injury.

Lastly, we did not find support for our last hypothesis because total scores on the EQ-i were not significantly related to, nor predictive of, ratings of positive, F (1, 28) = .92, p = .347; F (1, 54) = .09, p = .764, and negative stimuli, F (1, 28) = 1.45, p = .238; F (1, 54) = 1.63, p =.208, nor physiological activity (EDA) for the positive, F (1, 28) = .25, p = .622; F (1, 54) = .01, p = .933 , and negative, F (1, 28) = .18, p = .675; F (1, 54) = .04, p = .838, affective pictures for persons with and without self-reported MHI, respectively.

Discussion

The attenuated emotional arousal observed for persons with self-reported mild head trauma in this study is characteristic of persons with TBI (de Sousa et al., 2011; Hopkins et al., 2002). Overall, there was a general reduced physiological and self-reported emotional response for students with self-reported MHI despite increased reports of experiential stress relative to their peers with no history of head injury. This pattern replicates our prior research of *emotional underarousal* (Baker & Good, 2014b). Even persons with mild head trauma demonstrated altered emotional functioning in terms of decreased autonomic responses relative to those who did not report head injury and this profile is similar to persons with moderate/severe disruption, especially to the VMPFC region (Bechara et al., 2000; Hopkins et al., 2002; Tranel & Damasio, 1994).

Our primary interest in the current study was to examine whether history of head injury would differentiate other indices of emotional functioning via both self-report measures of components of social cognition (emotional intelligence, empathy), arousal, valence, intensity, and empathy ratings of emotionally-evocative stimuli, and autonomic affective sensitivity (i.e., EDA) to emotionally-evocative picture stimuli. As well, we examined if indices of emotional intelligence would be related to physiological emotional reactivity. Furthermore, we explored if emotional arousal state could be modified for persons with a history of head trauma for a longer period of time than previous arousal inductions (e.g., Baker & Good, 2014b).

Based on the limited evidence of examinations of impairments in components of social cognition after neural disruption for persons with moderate-to-severe injury (Spikman et al., 2012; Leopold et al., 2011), we expected that emotional intelligence

would vary between students with and without MHI. We did not find support for this hypothesis. However, in line with our hypothesis we found that students with MHI tended to report being less emotionally responsive to the distress of others. This pattern of behaviour for persons with MHI is similar to the common reports of unemotionality and a lack of concern for others from family members of persons with moderate/severe TBI (e.g., Milders et al., 2006). Furthermore, this component of emotional intelligence (i.e., responsivity to other's distress) may also be characteristic of empathic abilities (Decety & Jackson, 2004). Empathy may be defined not only as the ability to understand other's experiences, but also encompasses the ability to respond to other's distress (Decety & Jackson, 2004). These characteristics of empathic abilities have been argued to be comprised of cognitive and emotional components, respectively (e.g., Preston & de Waal, 2002). The trend observed in the current study may suggest that persons with MHI may be impaired with respect to the emotional, but not the cognitive, component of empathy. It is likely that the pattern of reduced self-reported ability to respond to the distress of others for persons with mild head trauma relative to their peers with no reported head trauma is a function of sustaining a head injury because empathic challenges are often a legacy of TBI (see Leopold et al., 2011; Spikman et al., 2012).

For instance, Wood and Williams (2008) have shown that persons with moderate/severe TBI reported experiencing low empathy (60% of TBI group vs. only 30% of controls reported low empathy). However, we did not find any significant differences between students with and without a history of MHI on the Toronto Empathy Questionnaire (Spreng et al., 2009), but this questionnaire does not compartmentalize the cognitive and emotional components of empathy. Further research should be conducted

with this population with possibly a different measure of empathy that distinguishes between these components. Moreover, a more comprehensive examination of social cognition in persons who have experienced head trauma is warranted. To our knowledge, only two studies have examined various aspects of social cognition with persons with severe neurocompromise (Leopold et al., 2011; Spikman et al., 2012). Leopold and colleagues (2011) demonstrated that persons with lesions to the VMPFC showed impairments in another aspect of social cognition, theory of mind, and that these deficits were related to measures of emotional intelligence and empathy. As such, an emerging body of research is demonstrating impairments in a variety of components of social-cognitive and socioemotional behaviours. Moreover, in the current study, students with MHI had significantly higher scores on Barchard's (2001) EI subscale of Positive Expressivity. Although we hypothesized that persons with head trauma would demonstrate attenuated emotional behaviours, this finding of increased positive affect is consistent with the positivity bias, a.k.a. a 'rose-coloured' view of self (Beer, 2007; Beer, Heerey, Keltner, Scabini, & Knight, 2003; Beer, John, Scabini, & Knight, 2006; 2010; Beer & Hughes, 2010; Beer, Lombardo, & Bhanji, 2010) and perhaps one's circumstances, also termed 'la belle indifference' (Pierre Janet [1849-1947]; see Iezzi, Duckworth, & Adams, 2004; Martelli, Nicholson, & Zasler, 2007).

Overall, both persons with and without a history of MHI demonstrated increased responses to the arousal manipulation via indices of self-report and physiological activation to the arousal manipulation (see Figures 2.2. and 2.3.). There were no significant 2-way interactions of either arousal measure across time by head injury history. Although, self-reported arousal to the emotional arousal induction did not vary

between students with and without a history of MHI, there was a significant main effect of MHI history such that students with MHI elicited attenuated EDA responses relative to their peers in response to the arousal manipulation, although they too demonstrated an increase in arousal post-manipulation that was sustained for a period of approximately 30 minutes. This finding differs from our previous study (Baker & Good, 2014b) in which modified arousal state was not maintained for greater than 15 minutes following a psychosocial stressor or relaxation experience for persons without MHI. The duration of the exposure to the affective stimuli was lengthy in the current study (~ 40 minutes) relative to our previous study (~ 10 minutes in Baker & Good, 2014) and may account for the maintenance of arousal state. Given the effective overall increase in physiological arousal of this manipulation we will utilize these stimuli (i.e., IAPS) in future studies to examine the relationship of arousal and cognitive performance (see Lupien et al., 2007) for persons with a history of head trauma.

Furthermore, and in concert with the underarousal hypothesis (see Baker & Good, 2014b), we expected that students with MHI relative to students with noMHI would provide lower ratings of arousal, valence, intensity and empathy for the affective picture stimuli (i.e., IAPS – Lang et al., 2008), but this hypothesis was not supported. However, and most interestingly, we found that EDA amplitude for students with self-reported MHI was similar for all stimuli types which did not support our hypothesis of reduced responding to negative stimuli (see de Sousa et al., 2012). Students with MHI were less responsive to the picture stimuli relative to their peers such that their EDA activity did not discriminate between the stimuli types (i.e., similar responses for the negative, positive, and ambiguous/neutral stimuli). In contrast, students without MHI produced

significantly different EDA responses to the negative, positive, and ambiguous/neutral stimuli and were most responsive to the negative stimuli, followed by the positive stimuli, and lastly produced smaller responses to the neutral/ambiguous stimuli. Although overall their EDA was relatively increased due to the exposure to the stimuli, across all stimuli types students with MHI elicited dampened physiological emotional responsivity relative to their peers without a history of MHI.

This finding is very similar to that found by de Sousa et al. (2012). In their study, participants with TBI (N = 64) or without TBI (N = 64) viewed pleasant and unpleasant film clips while facial electromyography and skin conductance responses were recorded. de Sousa and colleagues found that persons with TBI had reduced/limited frowning and smiling to both negative and pleasant videos, respectively, compared to persons without TBI. As well, skin conductance responses of persons with TBI were found to be significantly attenuated relative to the controls for the emotional stimuli (especially for the negative film clips). Another study by de Sousa et al. (2011) demonstrated that skin conductance reactivity of persons with TBI was similar to *both* negative and pleasant facial expression stimuli i.e., abnormal affective responsivity regardless of valence of stimuli. Although other research demonstrates that negative emotional responses are primarily impacted by TBI (e.g., Croker & McDonald, 2005; Dethier et al., 2013), de Sousa and colleagues (2011; 2012) have demonstrated blunted affective responsivity regardless of valence for persons with TBI and we have demonstrated that this pattern is also illustrated at the other end of the spectrum of injury severity for persons with mild head trauma (Alexander, 1995; Iverson & Lange, 2009).

Lastly, we did not find any significant relationships between emotional intelligence and EDA responsivity to the arousal manipulation as a function of a history of head injury. This examination was exploratory in nature especially with respect to the mild head trauma group. To our knowledge only one other study has examined this relationship for persons without a history of head trauma. EDA is an index of emotional arousal and can be used as a measure of emotional regulation (Benedeck & Kaerbach, 2010). Zysberg (2012) had hypothesized, as did we, that higher levels of EI would be associated with emotional regulation in terms of EDA responses (i.e., higher EI related to increased EDA response) to positive and negative images. Zysberg found that scores on an audio-visual test of emotional intelligence were significantly positively related to increased responsivity to the emotional stimuli. It is possible that we did not find a similar relationship in the current study due to the difference in study materials (i.e., a self-report measure of EI vs. an audio-visual measure [for this task the participants viewed short video clips of various socioemotional scenarios and identified others' emotions]). The EI measure used by Zysberg (2012) likely has better ecological validity.

A few limitations of the current study must be noted. One limitation is that the stimuli consisted of static images and were not dynamic (e.g., video clips). Static images are less realistic of everyday life interactions. As well, the self-report method of obtaining information regarding head trauma may be prone to challenges (e.g., bias, inaccuracy – e.g., Harrison, McLaughlin, & Coalter, 1996) and we did not have medical documentation regarding the head trauma event. However, and perhaps more strikingly, we have shown a pattern of socioemotional sequelae for persons with self-reported head injury that is similar to persons with moderate/severe TBI which illustrates a continuum

of injury severity (Alexander, 1995; Iverson & Lange, 2009). As well, students reported the head injury event to occur approximately 5 years earlier which suggests emotional sequelae postinjury is not likely transient (as in Ietswaart et al., 2008). Admittedly the direction of the relationship between mild head injury and emotional reactivity cannot be examined in the current cross-sectional study. It may indeed be possible that persons with lowered arousal are more likely to sustain head trauma.

In conclusion, the results of this study illustrate that even persons with self-reported 'mild' head trauma exhibit atypical affective responsivity to emotional stimuli in terms of physiological responses. As well, components of social cognition, particularly, emotional intelligence such as the ability to respond to others when distressed (trend) may be implicated in the emotional sequelae postinjury even for persons with MHI although further investigation is warranted. The implications of these findings include the possibility of persons with MHI showing emotional indifference or insensitivity to emotional happenings in the real world which may impact the quality of their social interactions with others as is seen for persons with moderate/severe TBI (e.g., Wood, Liossi, & Wood, 2005).

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STUDY 3: NEUROENDOCRINE AND AUTONOMIC INDICES OF STRESS RESPONSIVITY ACROSS THE SPECTRUM OF TRAUMATIC BRAIN INJURY SEVERITY[8]

Introduction

Emotional, cognitive, and behavioural changes are commonly observed following moderate or severe TBI (McAllister, 2011; Ponsford, 2013). Traumatic brain injury (TBI) commonly results in altered emotion regulation (e.g., Mathiesen, Forster, & Svendsen, 2004; Bornhofen & McDonald, 2008). The emotional changes such as flattened affect, emotional lability (e.g., outbursts) and irritability pose socioemotional challenges and may be attributed to 'personality' changes postinjury (Greve et al., 2001; Tateno, Jorge, & Robinson, 2003; Mathiesen et al., 2004). The experimental literature of persons with moderate or severe TBI has demonstrated impairments in emotion recognition ability (e.g., Ietswaart, Milders, Crawford, Currie, & Scott, 2008) and emotional reactivity post-injury (e.g., Hornak, Rolls, & Wade, 2006). Affective sensitivity has been shown to be variable in that persons with TBI are often emotionally labile (e.g., overly reactive) and/or demonstrate a reduced capacity for emotional experiences (e.g., Croker & McDonald, 2005; Hopkins, Dywan, & Segalowitz, 2002; Hornak et al., 1996). In the current study, we examined emotional volatility/lability or attenuated emotional responsiveness following TBI in terms of emotional arousal (both self-report and physiological indices) and overall emotional functioning (e.g., emotional intellect; affect recognition). Given that prior research has rather consistently demonstrated dampened emotional reactivity to emotional stimuli for persons with moderate to severe disruption to the frontal lobes (e.g., Damasio, Tranel, & Damasio, 1990; de Sousa et al., 2011;

[8] Portions of this study will be presented at the 91st Annual American Congress of Rehabilitation Medicine Conference: Progress in Rehabilitation Research, October, 2014, Toronto, Ontario.

closed head injury – Hopkins et al., 2002) and even self-reported mild head injury (Baker & Good, 2014) we tested the hypothesis of emotional dysregulation in terms of emotional underarousal (i.e., dampened reactivity) for persons with TBI across the spectrum of injury severity (mild, moderate/severe) via a novel investigation of indices of stress responsivity.

It is widely accepted that persons with moderate or severe disruption to the brain experience emotion regulation difficulties (e.g., Bornhofen & McDonald, 2008), commonly demonstrate a reduced capacity for emotional experiences (e.g., Hopkins et al., 2002; Hornak, Rolls, & Wade; 1996; Croker & McDonald, 2005), and an impaired ability to recognize emotional expressions (e.g., Bornhofen & McDonald, 2008; Dethier, Blairy, Rosenberg, & McDonald, 2013). More recent examinations have also demonstrated that impairments in aspects of social cognition such as empathy or theory of mind are also evident postinjury for persons with moderate or severe TBI (de Sousa et al., 2011; Leopold et al., 2012); however, emotional functioning following milder head trauma has not been explored to the same extent and the magnitude of the findings has been small (e.g., small effect size - see Panayiotou, Jackson, & Crowe, 2010).

Of particular note, the examination of stress responsivity postinjury has been limited to studies of post-traumatic stress disorder (e.g., Harvey & Bryant, 1998; Bryant & Harvey, 1999) or anxiety (e.g., Moore, Terryberry-Spohr, & Hope, 2006), especially for persons with mild head injury (but see Baker & Good, 2014a; Gouvier, Cubic, Jones, Brantley, & Cutlip, 1992). However, we have demonstrated that persons with a history of head trauma may have a lessened ability to interpret and respond to stressors/emotional events in what we have termed *emotional underarousal* (Baker & Good, 2010; 2014; St.

Cyr [Baker] & Good, 2007). Studies by Jung and Good (2007) and St.Cyr (Baker) and Good (2007; 2010; 2014) have shown that persons with self-reported mild head injury (i.e., trauma to the head sufficient to produce an altered state of consciousness – Kay et al., 1993; American Congress of Rehabilitation Medicine [ACRM]) that despite reporting increased life stressors (e.g., financial and relationship difficulties) were emotionally underaroused and may be less responsive to emotional stressors based on physiological indices (i.e., heart rate, electrodermal activation) and self-report measures of arousal state (e.g., anxiety). For example, we (Baker & Good, 2012) demonstrated that students with a history of mild head trauma elicited significantly reduced physiological responsivity (i.e., EDA amplitude) to emotionally-evocative stimuli (i.e., positive, negative, and ambiguous pictures) relative to those with no history of head trauma. We suggest that the emotional underarousal demonstrated by persons with moderate to severe disruption to the frontal lobes (e.g., Damasio et al., 1990; Hopkins et al., 2002; Naqvi, Shiv, & Bechara, 2004) follows a continuum of injury severity (Alexander, 1995; Iverson & Lange, 2009) because this pattern is illustrated even for persons with mild head trauma, albeit subtly (Baker & Good, 2014; Baker & Good, 2012). Furthermore, and in line with our findings, a study by Bay and colleagues (2009) reported that persons with mild to moderate TBI demonstrated hypocortisolemia (i.e., decreased salivary cortisol) and flattened diurnal patterns of cortisol. Bay et al.'s finding, in concert with our previous research (e.g., Baker & Good, 2014a) suggests that persons with neurological compromise (e.g., TBI) may demonstrate dysregulated stress responsivity.

A gap in the literature exists regarding emotional functioning of persons with TBI in terms of emotional arousal and stress responsivity. Similarly, affective difficulties,

such as the ability to identify others' emotions (Bornhofen & McDonald, 2008), has not been examined across the continuum of severity of injury (Alexander, 1995; Iverson & Lange, 2009 - i.e., more severe TBI, more emotional functioning difficulties). These issues were investigated in the current study.

A considerable amount of research has focused on understanding the regulation of the stress response (see Selye, 1936/1998) in both animal and human studies (e.g., McEwen, 2000; Sapolsky, Krey, & McEwen, 1986, Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; see McCormick, 2007 for discussion). In general, this work has emphasized the importance of the amygdala in initiating the stress response, its role in initiating activation of the hypothalamic-pituitary-adrenal (HPA) axis and its regulation involves hippocampal, amygdala activity (i.e., the bed nucleus of the stria terminalis), as well as the role of the ventromedial PFC in the perception of, and initiation of, sympathetic nervous system activity (e.g., Barbas, Saha, Rempel-Clower, & Ghashghaei, 2003; Diorio, Viaou, & Meaney, 1993; Sapolsky, Zola-Morgan, & Squire, 1991). Jaferi and Bhatnagar (2007) (and others) have discussed the role of the VMPFC and/or orbitofrontal cortex (OFC) and its influence on neuroendocrine and autonomic functions. In particular, the VMPFC/OFC region modulates stress-related behaviour/emotional arousal via its connectivity with subcortical regions (i.e., hypothalamus, amygdala - Amaral, Price, Pitkanen, & Carmichael, 1992; Barbas et al., 2003; Diorio et al., 1993; Jaferi & Bhatnagar, 2007). Activation of the HPA-axis in response to stressful stimuli (either 'external' or 'internal') and/or challenge to the system results in the release of cortisol (e.g., see McCormick, 2007 for discussion; Dickerson & Kemeny, 2004; Nicolson, 2008). Cortisol, a glucocorticoid, exerts widespread effects on the body (i.e.,

metabolism, immune response; see Fulford & Harbuz, 2005; Webster, Marketon, & Glaser, 2008) and ultimately assists in preparing the body to respond to the challenge be it real or perceived (Dikerson & Kemeny, 2004; Nicolson, 2008).

The glucocorticoid receptor ratio hypothesis has been proposed by de Kloet and colleagues (de Kloet, Karst, & Joels, 2008; de Kloet, 2000) and has similarities with the Yerkes-Dodson (1908) law with respect to curvilinear effects of glucocorticoids (Lupien et al., 2007). We have examined the influence of modified arousal state and its relationship to cognitive performance (see see Lupien et al., 2007 for review) for persons with mild head injury (e.g., Jung & Good, 2007; Baker & Good, 2010; 2014a; St.Cyr [Baker] & Good, 2007); however, we did not measure endocrine responses in these studies. Overall, in these studies we found some support for the underarousal hypothesis in that persons with MHI, in contrast to what is typically found (see Lupien et al., 2007 for review), showed cognitive benefits from being activated to a higher level of arousal via exposure to a psychosocial stressor or listening to music (e.g., memory tasks: St. Cyr & Good, 2007; attentional tasks: Jung & Good, 2007; decision-making tasks: Robb & Good, 2012). Further examination of the potential impact of modified arousal state on cognitive abilities is warranted and was investigated in the current study including measurement of salivary cortisol in response to an emotionally-charged stimuli/stressor.

Numerous studies have demonstrated changes in concentrations of salivary cortisol (e.g., Dickerson & Kemeny, 2004; Kirschbaum & Hellhammer, 2000) and salivary alpha-amylase (e.g., Nater & Rohleder, 2009) after challenge to the system in the form of stress paradigms such as psychosocial stress (see Dickerson & Kemeny, 2004) or exposure to emotionally evocative visual stimuli (e.g., viewing graphic videos or pictures – e.g.,

Ellenbogen, Schwartzman, Stewart, & Walker, 2002); however, to our knowledge, little, if any, research has examined these markers of stress responsivity for persons with a history of head trauma despite evidence of neuroendocrine disruption following moderate or severe TBI (e.g., post-traumatic hypopituitarism – see Agha & Thompson, 2006). Neuroendocrine dysfunction after moderate or severe TBI has been documented in both the acute postinjury phase (e.g., 7 days postinjury - Cernak, Savic, Lazarov, Joksimovic, & Markovic, 1999) and long-term recovery (e.g., one year postinjury - Krahulik, Zapletalova, Frysak, & Vaverka, 2010) in terms of indices of abnormal/disrupted pituitary function (e.g., Agha & Thompson, 2006). Similarly, atypical diurnal rhythms of cortisol have also been reported for persons who have sustained a TBI (e.g., see Bay et al., 2009). Cortisol follows a circadian rhythm and concentrations are highest early in the morning, and other than a slight rise after lunch, they steadily decrease throughout the day (see Kerkof, 1985 for discussion; Nicolson, 2008). Cortisol concentrations begin to rise prior to awakening and the cortisol awakening response (CAR) is characterized by peak concentrations in the first 30 to 40 minutes immediately following awakening (Clow, Hucklebridge, Stalder, Evans, & Thorne, 2010; Pruessner et al., 1997). The CAR has been implicated as being an index of one's ability to respond to stressors (Clow et al., 2010).

However, to our knowledge, limited research has investigated neuroendocrine indices of stress responsivity in the post-acute phase of TBI to examine potential hypo- or hyperarousal as well as possible atypical diurnal patterns of cortisol. We are only aware of one study that has examined indices of atypical or dysregulated stress responses as a function of head trauma (i.e., Bay et al., 2009) in which they found lower levels of

cortisol (an index of stress responsivity) and flat diurnal patterns in persons with mild and moderate TBI.

To gain insight into the emotional functioning of persons with a history of head trauma across the spectrum of injury severity in the current study, we examined responsivity to environmental and laboratory stressors for individuals as a function of severity of neurological compromise (no history of head trauma, mild head injury [MHI], moderate/severe TBI) via physiological (cortisol awakening response [CAR], cortisol responsivity, electrodermal activation [EDA], heart rate, and respiration) and self-reported indices. As suggested by Clow et al. (2010) and others, the CAR is a promising biomarker of emotional health, therefore examination of the CAR in the current study will provide potential insight into emotional dysregulation that is often experienced by persons with TBI. This is the first study to examine the CAR with this population as an index of ability to respond to the day's stressors. In addition, we also examined the effects of experimentally modified arousal state (e.g., viewing emotionally-evocative stimuli) on cognition as a function of history of neural disruption. We expected that increased arousal for those who are typically underaroused would improve cognitive performance (see Baker & Good, 2010; Jung & Good, 2007; Robb & Good, 2012; St. Cyr [Baker] & Good, 2007). This research is unique in that socioemotional functioning in terms of stress responses (self-report or physiological indices) is rarely examined in the TBI population; furthermore, this study is unique with respect to the emphasis on the continuum of the severity of brain injury (Alexander, 1995; Iverson & Lange, 2009). This is one of the only studies to examine: a) salivary cortisol responsivity to an experimental emotional induction and its potential impact on cognitive performance for persons with

head trauma; b) the cortisol awakening response in the TBI population; and, c) physiological (i.e, EDA, heart rate) and self-reported indices of arousal and emotional functioning (i.e., emotional intelligence – Baron, 1997; Barchard, 2001) as a function of injury severity (no head trauma, MHI, moderate/severe TBI).

Based on the prior, albeit scarce, research (e.g., Baker & Good, 2014; Bay et al., 2009), we expected to find evidence of dysregulated stress responsivity (e.g., lower levels of cortisol; electrodermal skin responses, and heart rate) and flattened patterns of cortisol (i.e., CAR) that would follow a gradient of severity of history of head trauma (i.e., those with more severe neural trauma would demonstrate even lower arousal/stress responses relative to those with moderate or mild injuries, although they, too will demonstrate attenuated responsivity compared to those with no history of head trauma). Furthermore, previous research from our lab has shown that cognitive performance on neuropsychological measures is enhanced for persons with (mild) head trauma when arousal levels are increased by experimental manipulation of arousal (Baker & Good, 2007; 2010; Jung & Good, 2007; Robb & Good, 2012). We expected to replicate this finding. This research will assist in answering gaps in the literature on TBI and will advance knowledge in the field of psychology regarding emotional functioning of persons with a history of head trauma and its potential impact on cognition and socioemotional interactions.

Hypotheses

Hypothesis 1. As in other studies (e.g., Baker & Good, 2014), we expected that persons with prior head injury would be emotionally underaroused as measured through both physiological and self-report measures, despite reporting increased life stressors

relative to persons without head trauma. We also hypothesized the emotional underarousal to follow a continuum of injury severity (i.e., greater injury severity, more dampened indices of arousal).

Hypothesis 2a. Indices of dysregulated arousal and stress response (i.e., CAR and salivary cortisol levels; physiological recordings of EDA, and heart rate) were hypothesized to differ as a function of injury severity (i.e., mild, moderate/severe) with persons with more severe trauma exhibiting more dysregulated arousal and less responsivity to the emotional arousal induction.

Hypothesis 2b. The study by Bay et al. (2009) documented flat diurnal patterns of cortisol for persons with mild and moderate TBI and therefore, in this exploratory study, we hypothesized a blunted CAR for persons with head trauma that would too, follow a gradient of injury severity.

Hypothesis 3. Induced-stress via exposure to emotionally-evocative stimuli was expected to heighten physiological arousal and, thereby, improve cognitive performance (e.g., neuropsychological test battery summary scores) for persons with compromised/lower physiological arousal and was expected to impair performance for persons with no history of head trauma.

Hypothesis 4. Components of emotional intelligence (and empathy) were expected to differ for persons with a history of head trauma in terms of attenuated emotional functioning for those with head trauma.

Hypothesis 5. Performance on an affect recognition task was expected to be lower for persons with head/brain injury relative to those with no head trauma. We also hypothesized that injury severity (no head trauma, MHI, moderate/severe) would predict

the ability to recognize emotions in others over and above emotional intelligence and psychiatric health.

Methods

Participants

Eighty-five participants ($N = 80$)[9] were recruited for participation from the university student population and the community for individual testing sessions held at Brock University. Participants were screened in a brief phone interview to ensure inclusion criteria were met (history of neurological compromise; matched-sample criteria; exclusion criteria: medication use, psychiatric or neurological disorder, shift work, poor sleep habits). Participants were also required to be fluent in English and to have normal or corrected vision. A brief introductory information session was conducted a few days prior to participation in the experimental session. Persons with no history of head trauma ($n = 40$; 20 females; 20 males), *Mage* = 20.73, *SD* = 3.02, mild head injury ($n = 32$; 17 females, 15 males), *Mage* = 20.78, *SD* = 2.37, and those with moderate/severe TBI ($n = 9$; 5 males, 4 females), *Mage* = 22.44, *SD* = 5.36, participated in this study (matched for age, education, and athletic history).

Materials

Self-report measures. As in other studies (i.e., Baker & Good, 2010; 2012; 2014), a variety of questionnaires were administered regarding health, social and emotional behaviours, and demographic information (i.e., Everyday Living Questionnaire [Baker & Good, 2008] head injury history; Life Stressors Scale (Holmes & Rahe, 1967 adapted by St.Cyr [Baker] & Good, 2007), etc.), symptom reporting (e.g., Postconcussive Syndrome

[9] Four participants were randomly selected to be removed from the data set for age and education matching in the sample. One participant discontinued the arousal task and this participant was excluded because he/she did not complete the remaining study protocol.

Checklist, PCSC; Gouvier et al., 1992), emotional responding (e.g., indices of state and trait anxiety [State Trait Anxiety Inventory, STAI; Spielberger, 1983]; indices of emotional intelligence [Emotional Quotient Inventory, EQ-i, Baron, 1997]), empathy [Toronto Empathy Questionnaire, Spreng et al., 2009]), personality characteristics, and psychiatric health (e.g., Symptom Assessment – 45, SA-45, Strategic Advantage; Beck Depression Inventory – II, BDI-II, Beck, 1997). These measures are described elsewhere (Baker & Good, 2012; 2014a) (*see p.111 of Study 2*). In addition, participants completed the Morningness-Eveningness Questionnaire (MEQ-SA; Horne & Ostberg, 1976) which was examined with respect to indices of the circadian rhythm of cortisol (see Kerkof, 1985 for discussion). The Life Orientation Test- Revised (LOT-R; Scheier, Carver, & Bridges, 1994) was also administered to provide an index of one's level of optimism (e.g., positivity bias). Participants also completed the Beck Depression Inventory – II[10] (BDI-II, Beck, 1987).

Morningness-Eveningness Questionnaire Self-Assessment Version (MEQ-SA; Horne & Ostberg, 1976). The MEQ is a 19-item questionnaire with four response options that provides an index of an individual's propensity and/or preference for activities during daytime or evening as well as sleep patterns (e.g., "What time would you get up if you were entirely free to plan your day?"). Responses are coded numerically and are combined to form a composite score. The composite score provides an index of the degree the individual favours morning versus evening. Scores range from 16 to 86.

[10] Study protocol maintained that any participants who demonstrated elevated scores on the BDI-II were contacted within 24 hours by the principal investigator(s) and provided with options for assistance (i.e., speak with a Psychologist, Campus Counselling Services). These participants (n = 5) were also contacted again 2 weeks later to follow-up. These participants acknowledged that they were already accessing services (i.e., campus services) and did not report an active episode.

Scores below 41 indicate 'evening types' whereas scores of 59 and above indicate 'morning types' and scores of 42-58 indicate 'intermediate types'.

Life Orientation Test-Revised (LOT-R; Scheier et al., 1994). The LOT-R provides a measure of one's level of optimism. Ten items (e.g., "In uncertain times, I usually expect the best") provide an index of optimistic view and are rated on a 4-point scale (*0* = strongly disagree, *1* = disagree, *2* = neutral, *3* = agree, *4* = strongly agree). Items 3, 7, and 9 are reversed coded and items 1, 3, 4, 7, 9 and 10 are tallied to provide a total score. Items 2, 5, 6, and 8 are filler items.

Neuropsychological measures. Measures of cognitive flexibility, attention, memory, and processing speed were used to assess cognitive functioning (i.e., selected subtests from the WAIS-IV, Weschler, 2009; WMS-IV, Weschler 2009; WRAT-4, Wilkinson & Robertson, 2006; and, DKEFS, Delis, Kaplan, & Kramer, 2001). Brief estimates of verbal and performance competence involved the Wording Reading subtest from the WRAT-4 and the Matrix Reasoning Subtest from the WAIS-IV. Subtests included: Trail Making Test (DKEFS), Letter-Number Sequencing (WMS-IV), Digit-Symbol Coding (WAIS-IV) and the Rey Complex Figure Test (Osterreith, 1944). The Affect Recognition subtest from the Social Cognition component of the Advanced Clinical Solutions Supplement to the WAIS-IV and WMS-IV (Weschler, 2009) was also administered[11]. See Appendix G for a list of neuropsychological measures and a detailed description.

[11] Note that the Iowa Gambling Task (IGT; Bechara et al., 1994) was also used in the study protocol as a decision-making task, but data from this measure form part of another study.

Arousal state measures. Self-reported arousal state (i.e., rate current arousal state on scale of 1 *very relaxed* to 10 *very stressed*), physiological indices of arousal and stress responding (i.e., EDA, heart rate, respiration, salivary cortisol) were obtained.

Verbal self-report of arousal state. Self-reported arousal state was measured by having the participant rate his/her current arousal state on scale of 1 *very relaxed* to 10 *very stressed*.

Psychophysiological arousal measures. Physiological activity during the testing session was recorded via Polygraph Professional (Limestone Technologies, 2008) equipment (as described in Baker & Good, 2014) using the Datapac USB™ 16-bit Data Acquisition Instrument with accompanying software. EDA, heart rate, and respiration data were collected. EDA was recorded via silver-silver chloride plated pads placed on the index and fourth fingers of the non-dominant hand. Electrodermal responses were measured in amplitude (i.e., the height of the electrodermal response measured in microsiemens [µ*S*]). Heart rate was recorded via a pulse oximeter on the middle finger of the non-dominant hand and was measured in beats per minute (*bpm*). Respiration was recorded via pneumatic bands placed at the level of the sternum and the abdomen. Respiration was measured in cycles per minute (*cpm*), but was not a primary variable of interest for this study. All data were carefully screened and inspected manually for artifact prior to analysis.

Salivary cortisol. Six saliva samples were collected from each participant: the evening before the testing session, immediately upon awakening on day of testing session and 45 minutes later[12], and pre-post arousal manipulation to examine stress responsivity (as well as diurnal rhythms). The CAR was measured as a comparison of the mean level

[12] See Clow et al. (2010) regarding measurement of the CAR.

of salivary cortisol from first morning sample, which was provided immediately upon awakening, to the second morning sample which was provided approximately 45 minutes later (see Clow et al., 2010).

Emotional arousal induction. The International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2005; 2008) are emotionally-evocative stimuli developed by the NIMH Center for Emotion and Attention (CSEA) at the University of Florida. We utilized a subset of 123 of these stimuli that were previously used in another study (Baker & Good, 2012) as they were found to induce increased physiological emotional arousal. In Baker and Good (2012), the picture stimuli were originally selected from the IAPS (Lang et al., 2005; 2008) based on high valence and high arousal ratings from previous studies' normative data (Calvo & Avero, 2009; Lang et al., 2008; Libkuman, Otani, Kern, Viger, & Novak, 2007). In the current study, participants viewed 45 pictures that were randomly presented (15 negative/unpleasant images, 15 positive/pleasant images, and 15 neutral/ambiguous) over approximately 15 minutes. Participants rated the pictures on 4 Likert scales (arousal and valence scales are derived from the Self-Assessment Manikin (SAM; Bradley & Lang, 1994): arousal (low/calm to high/excited), valence/pleasure (unpleasant/unhappy to pleasant/happy), intensity (not intense to extremely intense), and empathy (no empathy to significant empathy). These 45 images were selected based on responses of a pilot study of 40 participants – images (negative, positive) that were most arousing and those that were most neutral/ambiguous as indicated by EDA amplitude were selected. Scenes involving erotica were excluded.

Salivary Cortisol Immunoassays. Salivary cortisol samples (6 samples per participant: evening/bedtime, immediately upon awakening, 2nd morning sample; upon

arrival to testing session, pre – and – post arousal induction – see Figure 3.1) were collected in 5 ml polystyrene tubes (http://www.simport.com/products/clinical/tubes/t405-t406-cultubes.html) and were stored in a freezer at -20 degrees until assayed with Salivary Cortisol Enzyme Immunoassay kits (SLV 2930) from DRG International, Inc. (Mountainside, New Jersey). Frozen saliva samples were thawed and centrifuged at 1730 g for 15 minutes then a 100 *uL* of each sample was dispensed via pipette into microtiterwells. Optical densities were determined via a plate reader set at ±450 *nm* (Bio-tek Synergy). A double determination process was used in that all saliva samples were measured twice[13] and the intra-assay coefficients of variation (CV) were less than 10%. The CV is basically an index of the relative intra-assay performance (DRG International User's Manual). If the CV criterion was not met across the duplicate measure of a single sample, the sample was re-assayed to provide another comparison. DRG reported the intra-assay and inter-assay CVs to be 2.65% and 6.64%, on average, respectively, for the salivary cortisol enzyme assays. All assays were conducted by the Developmental Neuroendocrinology Laboratory at Brock University, Canada (Dr. Cheryl McCormick). The first author was involved with the assay procedure and all analyses. The mean salivary cortisol measure is provided in ng/mL with average adult levels ranging between 1.2 ng/mL to 14.7 ng/mL (DRG International User's Manual).

Procedures

[13] Note. Two participants provided an insufficient quantity of saliva (for one sample each) to complete the double determination process, therefore only a single reading was provided. One sample was contaminated and therefore the assay was not conducted (participant #118). Further, one participant discontinued the arousal manipulation task therefore all endocrine measures were not appropriate for this participant (samples 5 & 6) – this participant was excluded from analyses.

Participants were recruited from Brock University, the Ontario Brain Injury Association, and local neuropsychologist's offices. Advertisements described the study as examining *Emotion and Cognition* and made no mention of an interest in brain injury to avoid diagnosis threat (Suhr & Gunstad, 2002). Participants were naïve to the purpose of the study. Participants were screened via a brief phone interview to ensure inclusion criteria were met (i.e., history of neurological compromise; matched-sample criteria). Verbal informed consent was obtained during the telephone interview regarding the provision of demographic information and health-related characteristics. Participants were matched on demographic factors, primarily history of head injury, age, sex, and level of education, as well as exercise/athletic history. Exclusion criteria included being older than 35 years of age, taking prescription medication that potentially interferes with the measurement of salivary cortisol (e.g., corticosteroids, thyroid medication, etc.), neurological disorders (e.g., multiple sclerosis), shift work, and poor/irregular sleep patterns. Participants were also required to be fluent in English and have normal/corrected vision. Eligible participants were then invited to the testing facilities at Brock University for an introductory session. In the introductory meeting, participants were provided with written informed consent and further information regarding study participation (see Appendix G). Instructions and materials to collect evening and morning saliva samples were provided to the participant and they were instructed to bring them with her/him to the testing session. All testing sessions occurred during the hours of 11:00 (a.m.) to 17:00 (p.m).

For the testing session, participants were connected to physiological recording equipment for recordings of EDA, heart rate, and respiration (via Polygraph Professional

Suite; Limestone Technologies Inc., 2008). For baseline physiological measures of arousal, participants were asked to provide another salivary sample and then physiological recordings were taken for a 3-minute baseline period. Participants then completed brief subtests from neuropsychological test batteries as pre-manipulation measures of cognitive flexibility, attention, memory, and processing speed (i.e., select subtests from the WAIS-IV, Weschler, 2009; WMS-IV, Weschler, 2009; DKEFS, Kaplan et al., 2001; WRAT-4, Wilkinson & Roberston, 2006; Advanced Clinical Solutions Supplement: Social Cognition to the WAIS-IV and WMS-IV, Weschler, 2009).

Participants were then exposed to an experimental manipulation of emotional arousal (i.e., a subset of the IAPS stimuli [Lang et al., 2008] - 15 positive, 15 negative, and 15 ambiguous/neutral pictures used in the emotional arousal induction in Baker & Good, 2012) and provided ratings of arousal, valence, intensity, and empathy for each picture. Physiological recordings of EDA, heart rate, and respiration were obtained throughout via Polygraph Professional (2008) software. Post-manipulation, participants were asked to provide another saliva sample[14]. Participants then completed post-manipulation neuropsychological testing to assess changes in cognition as a function of manipulated arousal state. Note that the alternate versions of the neuropsychological tests that were used pre- and post arousal manipulation were administered to avoid practice effects. Interspersed throughout the testing session participants provided self-reports of arousal state (i.e., on a scale of 1 *relaxed* to 10 *stressed*). Lastly, participants completed a variety of questionnaires examining health and social behaviours, demographic information (i.e., head injury history, life stressors, etc.), symptom reporting (e.g., PCSC; Gouvier et al., 1992), emotional responding (e.g., indices of state and trait anxiety [State

[14] As recommended in Dickerson and Kemeny's (2004) review, salivary cortisol is best measured at least 30 minutes from onset of a stressor.

Trait Anxiety Inventory; STAI; Spielberger, 1983]; indices of emotional intelligence [BarOn Emotional Quotient Inventory; EQ-i, Baron, 1997; Emotional Intelligence questionnaire; EI, Barchard, 2001]), and personality characteristics described elsewhere (see p. 111). A final recording of physiological arousal was taken at the end of the testing session and participants were disconnected from the equipment and debriefed as to the nature of the study. Participants were naive as to the purpose of the study until the debriefing session[15]. Participants had the opportunity to receive research participation hours for applicable courses at the university or receive a small monetary honorarium ($15). Salivary samples were stored in a freezer at -20 degrees until enzyme-linked immunoassays were performed. This research received clearance from the local university's Research Ethics Board (BREB application Good 12-084) (see Appendix F) and was awarded a Brock University Social Sciences and Humanities Research Council of Canada Institutional Grant (BSIG Start Date: January 2013).

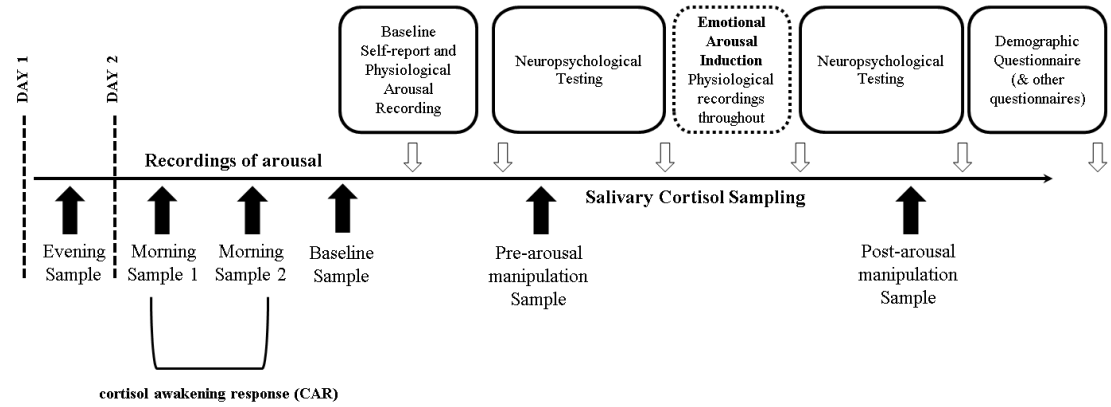

Figure 3.1. Emotion and Cognition Study protocol.

[15] To avoid 'diagnosis threat' (Suhr & Gunstad, 2002) and/or bias and expectations of performance, the study was advertised as an *Emotion and Cognition Study* with no mention of head injury as a variable of interest until debriefing session.

Data analysis

This study is a quasi-experimental mixed-model design (group variable is severity of TBI: no history of head trama, mild head injury, moderate/severe TBI). Pearson Chi-Square statistic and one-way analyses of variance (ANOVAs) were used to compare demographic information between students with and without a history of head injury/TBI. To examine the underarousal hypothesis, baseline arousal (physiological [i.e., EDA] and/or self-reported indices) was examined as a function of injury severity (noMHI, MHI, moderate/severe TBI) via one-way ANOVAs. Mixed model ANOVAs were conducted for physiological (i.e., EDA, HR, and salivary cortisol) and self-reported measures of arousal to examine differences between head trauma groups and/or responsivity across the testing session and diurnal patterns (especially the CAR).

Cognitive performance on brief estimates of intellectual functioning (verbal, performance, affective) at baseline (i.e., prior to any emotional arousal induction) was examined via one-way ANOVAs across severity groups. Mixed model 3 (Severity of Injury: noMHI, MHI, moderate/severe TBI) X 2 (time: pre, post emotional arousal induction) ANOVAs were conducted to examine the potential influence of the arousal manipulation on cognitive performance. Lastly, we tested the hypothesis of dysregulated stress responses via the cortisol awakening response via repeated measures ANOVAs for each group (noMHI, MHI, and moderate/severe TBI).

Overall emotional functioning as indexed by psychiatric health (i.e., SA-45), emotional intelligence measures (i.e., EQ-I), and empathic ability (i.e., TEQ) was examined for group differences (i.e., lessened emotional responding) via ANOVAs across injury severity groups. Emotional intelligence was analyzed for potential influence

in emotional arousal state and/or responsivity via regression analysis. We conducted hierarchical multiple regression analysis to examine if severity of injury (no head trauma, MHI, moderate/severe) could predict performance on a task involving identifying the emotional expressions of others (ACS - Affect Recognition) over and above emotional intelligence and psychiatric health. All data analysis was performed with SPSS for Windows Version 18.0 (SPSS Inc., 2008) and Polygraph Professional (2008). Immunoassays were conducted for salivary cortisol as indicated above.

Results

Demographics

See Table 3.1 for a comparison of indicators of severity of TBI. All of the students who reported a LOC associated with their injuries in the MHI group (55.23%, *n* = 17) experienced the LOC for under 30 minutes in duration and therefore meet criteria for 'mild' head injury (ACRM; Kay et al., 1993). The majority of students with self-reported MHI (88.2%, *n* = 15) experienced the LOC for less than 5 minutes, demonstrating these students are at the very 'mild' end of the continuum of injury severity (Alexander, 1995; Iverson & Lange, 2009). On the other hand, the majority of students with moderate/severe TBI experienced a LOC (77.8%; *n* = 7). Students with MHI reported their injury to have occurred on average 8.49 years earlier (*SD* = 7.74) at approximately 13 years of age (*SD* = 5.09) whereas those with moderate/severe TBI had experienced their injury more recently, 3.33 years earlier, *SD* = 3.27, occurring at approximately 19 years of age. Not surprisingly, more students with moderate/severe TBI received medical treatment for their injury than those with MHI. The majority of students with MHI reported sports-related injuries (*N* = 18; 56%) as the primary cause of head injury,

followed by injuries occurring from falls (N = 9; 28%), and other causes (e.g., fighting; N = 4; 13%) with one student incurring injury via a motor vehicle collision (MVC) (see Figures 3.2 and 3.3). Fifteen percent (N = 5) of students reported more than one MHI. In contrast, MVCs were the primary cause of injury for students with moderate/severe TBI (N = 5; 56%) followed by sports related activities, and falls. Students with MHI and moderate/severe TBI did not differ in age from students with no history of head trauma, F (2, 78) = 1.18, p = .313. Years of education was similarly represented across the groups, χ^2 (3) = 4.12, p = .242. Students were also matched for athletic status (i.e., regular exercise, participation in organized sports).

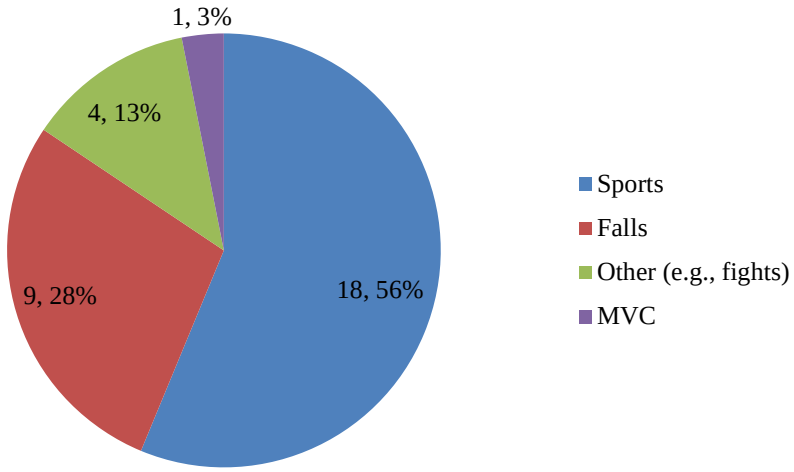

Figure 3.2. Etiology of mild head injuries.

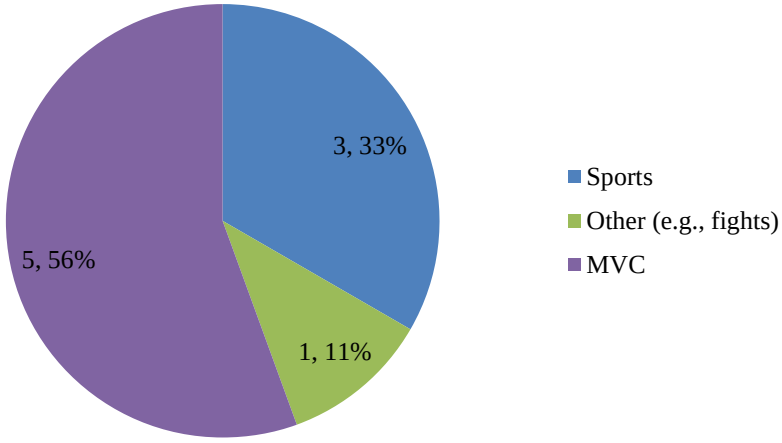

Figure 3.3. Etiology of moderate/severe TBIs.

Table 3.1. *Indicators of TBI Severity.*

	n = 32		*n* = 9	
	Mild Head Injury		*Moderate/Severe TBI*	
Mean age at injury	13.56 (5.09)		22.44 (5.36)	
Mean years since injury	8.49 (7.74)		3.33 (3.27)	
	n	Percentage	*n*	Percentage
Experienced symptoms for >20 minutes	13	40.60	9	100
Concussion	18	56.30	9	100
Received medical treatment	13	40.60	8	88.90
Stitches	3	9.40	1	11.10
Overnight stay at medical facility	1	3.10	5	55.60
Altered State of Consciousness	15	46.87	2	22.20
Loss of Consciousness	17	53.13	7	77.80
< 5 minutes	15	88.20	2	28.60
< 30 minutes	2	11.80	2	28.60
>24 hours	0	0	3	42.90

Note. Numbers in parentheses are standard deviations.

Baseline Cognitive Testing

Students with MHI and students with moderate/severe TBI did not differ from students with no history of head trauma on brief estimates of intellect in terms of verbal competence (i.e., WRAT-4 Word Reading Total Score), F (2, 78) = 1.55, p = .218, and performance ability (i.e., Matrix Reasoning Total Score from WAIS-IV), F (2, 78) = 1.05, p = .356. In line with our hypothesis, a trend was observed for scores on the Affect Recognition task (ACS – WAIS-IV), F (2, 78) = 2.88, p = .062, in that students with moderate/severe injury were worse at identifying facial expressions of emotion relative to those with no reported head trauma, *Least Significant Difference* [*LSD*], p = .026. Performance on the affect recognition task was not significantly different between students with MHI to students with no head trauma, *LSD*, p = .169, nor when compared to those with moderate/severe TBI, p = .182. Although the pattern of affective ability was in the expected direction, it did not reach statistical convention (refer to Figure 3.4). Refer to Table 3.2 for descriptive statistics.

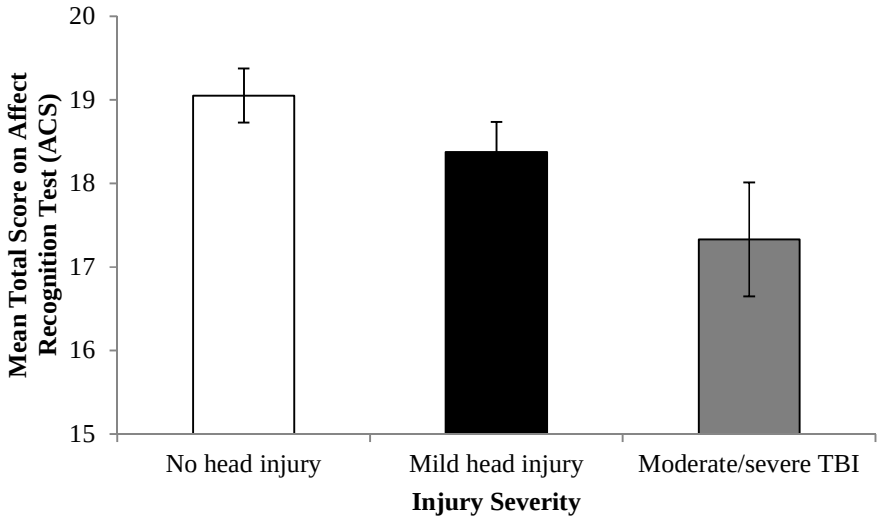

Figure 3.4. Affect Recognition Total Scores across injury severity groups.

Table 3.2

Performance on Neuropsychological Measures across History of Head Trauma Groups

	No Head Injury *N* = 40		Mild Head Injury *N* = 32		Moderate/Severe TBI *N* = 9	
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*
WRAT-4 Word Reading Score	47.33	4.32	46.72	4.25	50.00	8.78
WRAT-4 Word Reading Completion Time (seconds)	73.87	24.75	73.13	17.07	77.45	20.21
WAIS-IV Matrix Reasoning	16.98	4.25	17.06	3.88	19.11	4.23
Affect Recognition** (ACS WAIS-IV/WMS-IV)	19.05	2.01	18.37	2.12	17.33	1.94

** $p < .10$

Stress Responsivity

Arousal.

At rest, before any emotional arousal manipulation, students who had a history of head/brain injury demonstrated significantly dampened resting EDA responses relative to students who reported no history of head injury, $F(2, 78) = 61.08$, $p < .001$ (see Figure 3.5), but comparisons demonstrated no difference in EDA amplitude between students with MHI and those with moderate/severe TBI – both had reduced responding, *LSD* $p = .327$ (refer to Figure 3.5). Similarly, this pattern of underarousal was evidenced via resting heart rate (*bpm*) as a function of injury severity such that students with no history of head injury had significantly higher heart rate (*bpm*), and those with MHI elicited significantly lower *bpm*, followed by those moderate/severe TBI demonstrating the lowest resting heart rate, $F(2, 78) = 3.66$, $p = .030$ (all *LSD* multiple comparisons significant *p*s < .05) (refer to Figure 3.6) . Although following a similar pattern to previous research (Baker & Good, 2014), there was no significant difference among students with noMHI ($M = 95.85$, $SD = 68.90$), MHI ($M = 108.25$ $SD = 74.38$) and moderate/severe TBI ($M = 118.89$, $SD = 49.11$) for the number of life stressors they reported experiencing, F (2, 78) = .54, $p = .585$, nor their self-reported arousal state, F (2, 78), 1.93, $p = .151$.

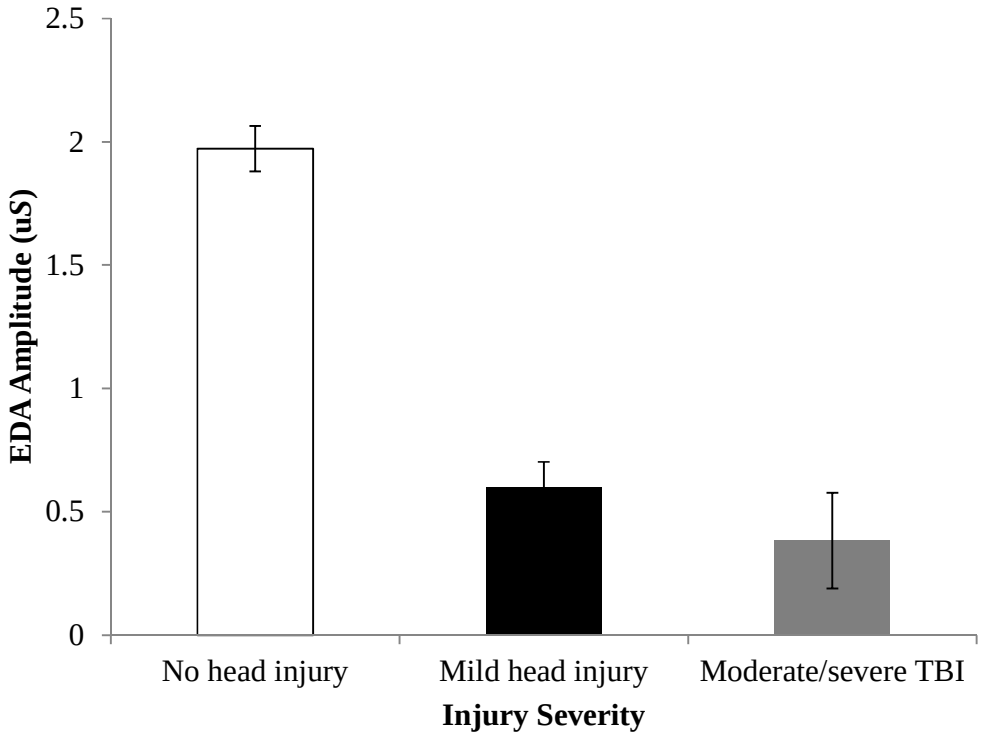

Figure 3.5. Mean resting EDA amplitude between injury severity groups.

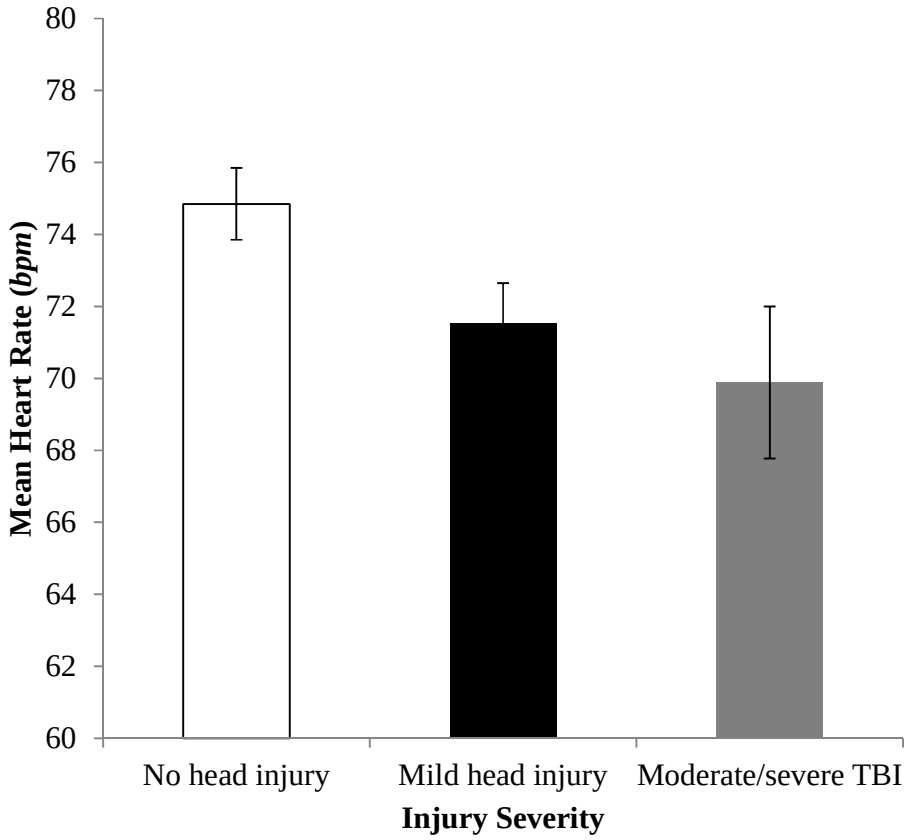

Figure 3.6. Mean resting heart rate (*bpm*) as a function of injury severity.

Reactivity to the emotional arousal induction across the testing session.

Mean self-reported arousal state across the testing session in response to the emotional arousal induction. A 3 (severity of injury: no MHI, MHI, moderate/severe TBI) by 3 (time: baseline, post emotional arousal induction, end of session) mixed model ANOVA demonstrated that self-reported emotional arousal state (1 *relaxed* to 10 *stressed*) significantly increased in response to the emotional arousal induction, F (2, 156) = 75.54, $p < .001$, *LSD p*s < .05 (refer to Figure 3.7). There was a trend for an interaction of these variables such that students with moderate/severe TBI rated their

emotional arousal significantly higher than the other two groups after exposure to the emotionally-evocative stimuli, F (4, 156) = 2.06, p = .088.

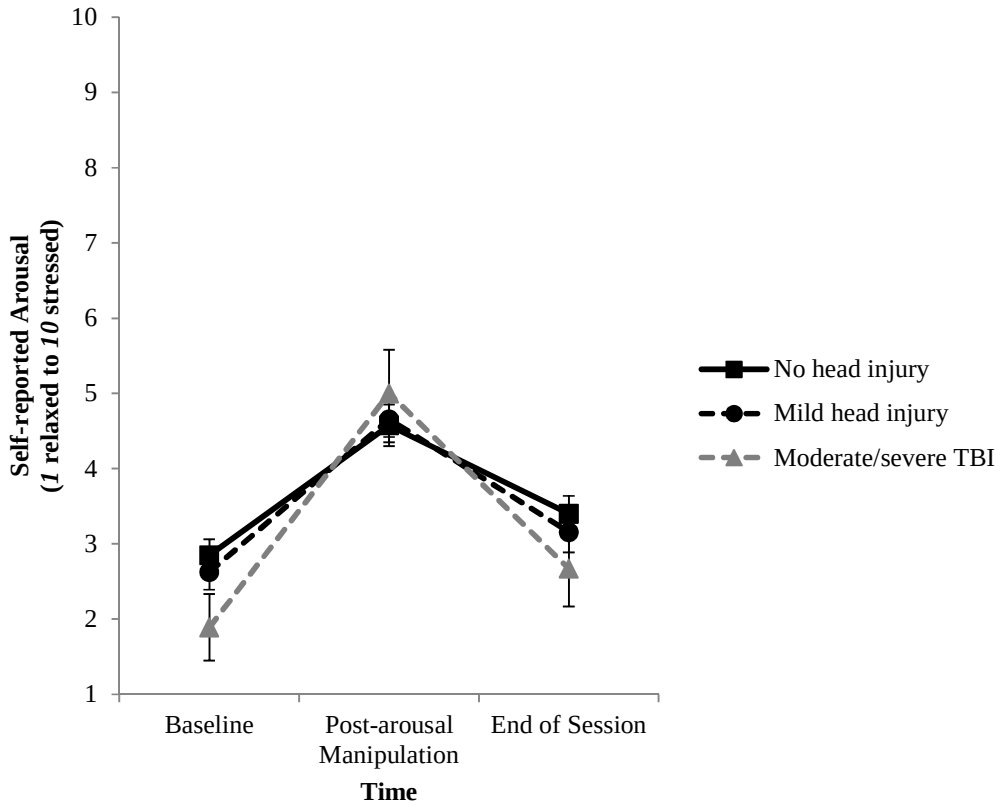

Figure 3.7. Mean self-reported arousal state across the testing session in response to the emotional arousal induction among injury severity groups.

Mean EDA responsivity to the emotional arousal induction. A 3 (severity of injury: no MHI, MHI, moderate/severe TBI) by 3 (time: baseline, post emotional arousal manipulation, end of session) mixed model ANOVA demonstrated that EDA amplitude significantly increased in response to the stressor, F (2, 156) = 19.56, p < .001 (multiple comparisons significant for all time periods *LSD p*s < .05). There was a significant main effect of injury severity, F (2, 78) = 57.59, p < .001, such that students with MHI and

moderate/severe TBI produced significantly smaller EDA amplitude than students who had not experienced a head injury, *p*s < .05, but both injury groups (MHI, moderate/severe TBI) demonstrated similar attenuated responsivity, *LSD p* = .335. There was no significant interaction, *F* (4, 156) = .71, *p* = .585 (see Figure 3.8).

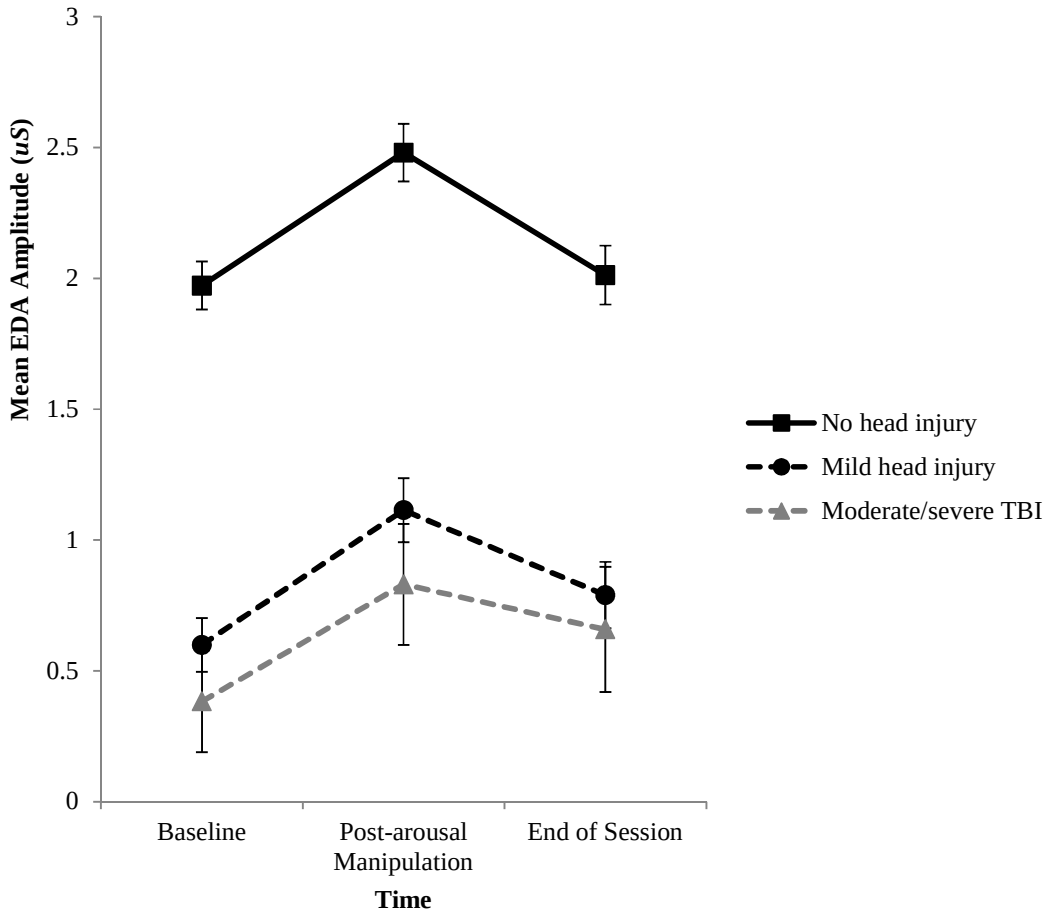

Figure 3.8. Mean EDA amplitude in response to the emotional arousal induction across injury severity groups.

Mean heart rate activity in response to the emotional arousal manipulation.

A 3 (severity of injury: no MHI, MHI, moderate/severe TBI) by 3 (time: baseline, post emotional arousal manipulation, end of testing session) mixed model ANOVA demonstrated that the average number of beats per minute significantly increased in response to the stressor, F 2, 156) = 49.27, $p < .001$, but there was no significant main effect of injury severity, F (2, 78) = 1.91, $p = .156$, nor a significant interaction observed, F (4, 156) = 1.25, $p = .290$ (see Figure 3.9).

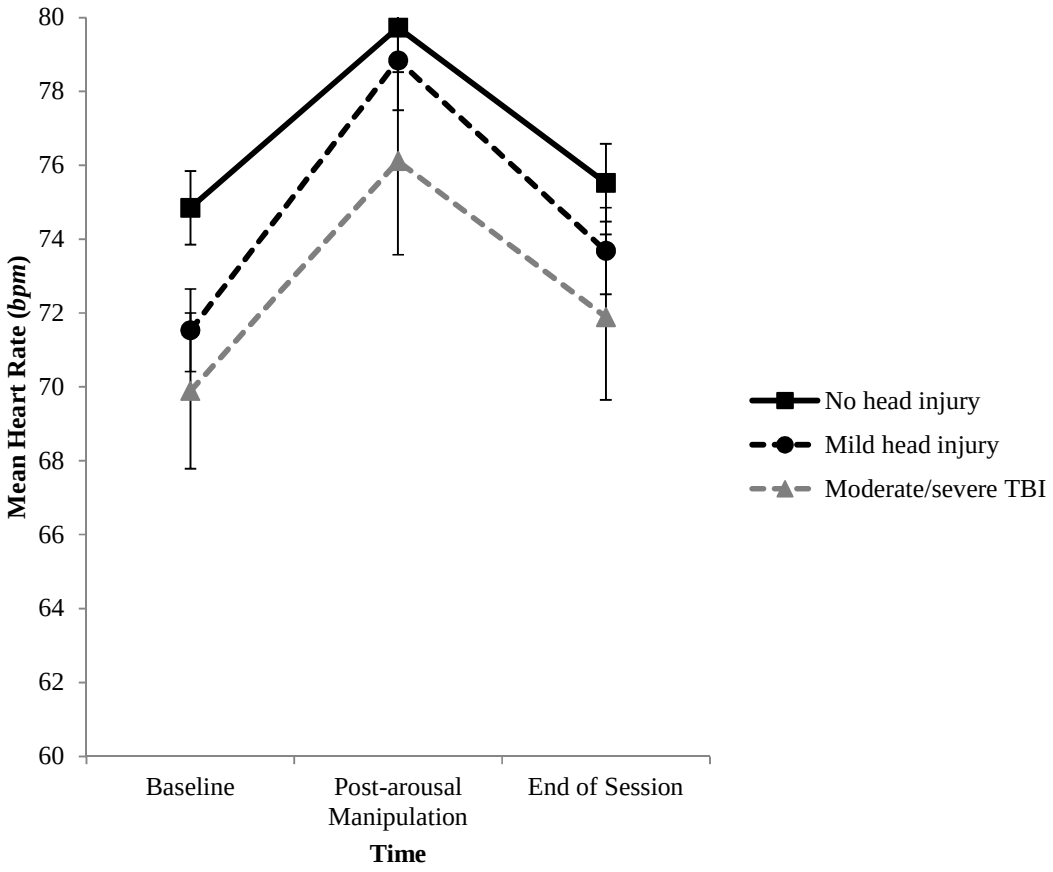

Figure 3.9. Mean heart rate (*bpm*) in response to the arousal manipulation across injury severity groups.

Mean Salivary Cortisol across the testing session. We examined another index of stress responsivity, mean salivary cortisol, via a 3 (severity of injury: no MHI, MHI, moderate/severe TBI) X 3 (time: baseline, pre, and post emotional arousal manipulation) mixed model ANOVA. In contrast to our hypothesis, there was a significant main effect of time such that mean salivary cortisol (*ng/mL*) decreased across the testing session, F (1.31, 100.84) = 31.92, $p < .001$, and did not increase in response to the stressor, *LSD p*s > .05. There was a trend for students with moderate/severe TBI to have higher average salivary cortisol, F (2, 77) = 2.50, $p = .089$.There was a trend for an interaction, F (262, 100.84) = 2.36, $p = .085$.

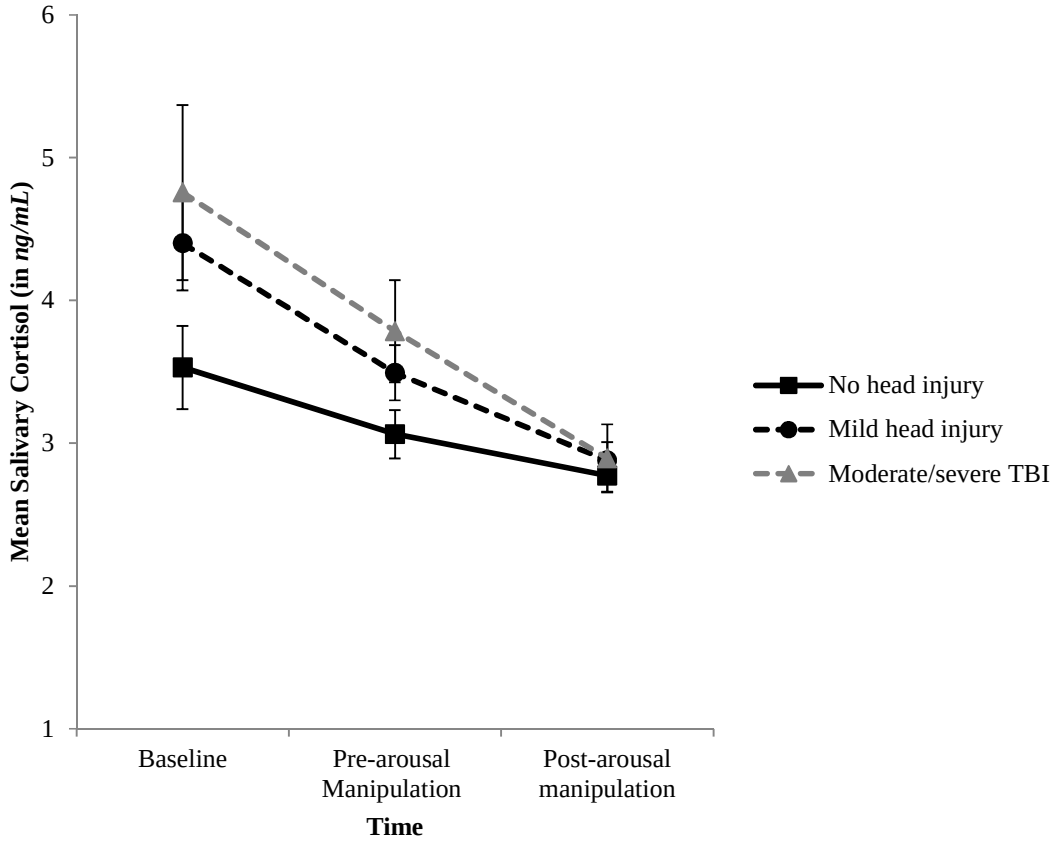

Figure 3.10. Mean salivary cortisol across the testing session.

Pre-post Emotional Arousal Manipulation Cognitive Testing

We tested the theory that persons with injury to the head/brain are underaroused. Based on the Yerkes-Dodson relationship between arousal and performance (1908; also see Lupien et al., 2007 for effects on cognition) and our previous data (e.g., St. Cyr [Baker] & Good, 2007), we expected them to perform better on cognitive tasks after exposure to emotionally-evocative task (i.e., IAPS stimuli), whereas persons without reported head trauma were expected to perform worse on cognitive tasks post-emotional arousal manipulation. We conducted 3 X 2 mixed model ANOVAs with severity of injury (noMHI, MHI, moderate/severe TBI) as the between-subjects factor and pre-post scores on the cognitive tasks as the repeated measure. There was very limited support for this hypothesis as discussed for the following measures (see Table 3.3).

Visuospatial memory. Students performed worse on the Rey Complex Figure task over time, F (1, 78) = 9.95, p = .002, and visuospatial memory abilities did not differ amongst the injury severity groups, F (2, 78) = 1.25, p = .292, nor as a function of time by severity of injury, F (2, 78) = .35, p = .706.

Digit Symbol Coding. The 3 X 2 ANOVA did not demonstrate any significant differences between the head trauma groups as a function of time (pre-post emotional arousal manipulation) on the Digit Symbol Coding task, F (2, 78) = 2.58, p = .082, but students performed worse on this task when repeated, F (1, 78) = 5.56, p = .021. There was no significant difference among the injury severity groups for this attentional task, F (2, 78) = .97, p = .384.

Letter-number Sequencing. Students performed significantly better on this working memory and attentional task with repeated testing, F (1, 78) = 41.97, p < .001,

and there was a significant 2-way interaction with severity of injury, F (2, 78) = 12.19, p < .001. Separate repeated measures ANOVAs for each severity of injury group revealed that students with mild head injury, F (1, 31) = 25.00, p < .001, and moderate/severe TBI, F (1, 8) = 72.00, p < .001, performed significantly better on this task with exposure to the emotional arousal induction in contrast to students without a history of head trauma who did not change across pre-post emotional arousal manipulation, F (1, 39) = .27, p = .609.

Trails. Students performed faster on the Trails task with repeated testing, F (1, 78) = 12.15, p < .001, but did not differ in performance on this task among injury severity groups, F (2, 78) = .63, p = .539, nor did these produce a significant interaction, F (2, 78) = 2.37, p = .100.

Table 3.3.

Performance on Neuropsychological Measures across History of Head Trauma Groups Pre- and – post Emotional Arousal Manipulation

	No Head Injury $N = 40$		Mild Head Injury $N = 32$		Moderate/Severe TBI $N = 9$	
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*
Digit Symbol Coding	83.18	13.08	83.44	16.61	75.33	19.05
Pre-manipulation						
Post-manipulation	77.78	11.34	81.82	17.68	74.67	19.09
Letter Number Sequencing*	11.40	2.01	10.09	2.31	9.33	1.73
Pre-manipulation						
Post-manipulation	11.60	3.00	12.59	2.61	13.33	1.94
Trail Making Test	34.56	10.16	36.11	12.61	42.01	14.36
Pre-manipulation						
Post-manipulation	33.46	10.42	31.70	12.73	34.24	10.23
Rey Complex Figure	24.10	5.16	22.28	4.76	22.00	7.09
Pre-manipulation						
Post-manipulation	22.86	5.65	21.00	4.59	21.44	6.89

*$p < .05$

Cortisol Awakening Response

We tested the underarousal hypothesis in terms of emotional/stress dysregulation as indexed by CAR. We hypothesized that persons with MHI and moderate/severe TBI would demonstrate a blunted CAR response and that the attenuated response would follow a gradient of severity of injury (see Figure 3.11).

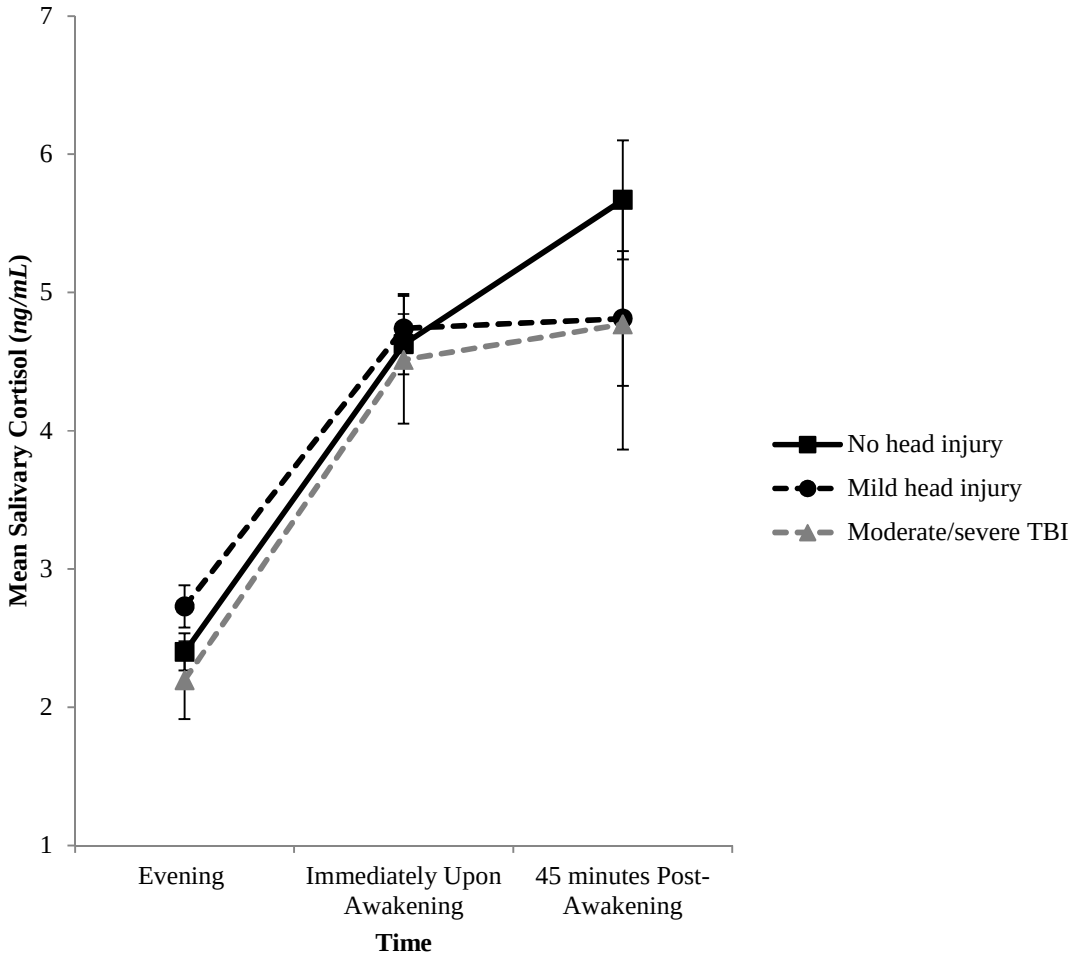

Figure 3.11. Blunted Cortisol Awakening Response for students with mild head injury and moderate/severe TBI relative to students with no history of head injury.

As illustrated above in Figure 3.11, students demonstrated significantly lower average salivary cortisol concentrations in the evening than in the morning as to be expected; however, upon awakening repeated measure analysis revealed that students with no reported head trauma illustrated the typical increase 45 minutes after waking i.e., CAR (from 4.62 ng/mL [1.16] to 5.67 ng/mL [3.40]), F (1, 39) = 3.85, p = .057. As hypothesized, students with self-reported mild head trauma did not produce a significant increase in salivary cortisol (4.74 ng/mL [1.67] to 4.81 ng/mL [1.88]), F (1, 30) = .03, p =.875, and students with moderate/severe TBI also did not demonstrate a typical CAR, (4.51 ng/mL [1.26] to 4.77 ng/mL [1.16]), F (1, 8) = .21, p =.657. Students who had a history of either mild head trauma or moderate/severe TBI demonstrated a blunted CAR.

Other Indices of Emotional Responsivity

Students did not differ as a function of a history of head trauma/brain injury across clinical and standardized indices of psychological health. In particular, students did not demonstrate significantly different scores on the Global Severity Index of the SA-45 (Strategic Advantage, 1998), F (2, 78) = .85, p = .432, nor its subscales (e.g., depression, paranoid ideations, psychoticism), ps > .05. Similarly they did not differ on measures of state, F (2, 78) = .07, p = .930, or trait anxiety (STAI, Spielberger, 1983), F (2, 78) = 1.23, p = .298, as a function of severity of injury. As well, clinical indices of depression (i.e., BDI-II, Beck, 1997) did not differ amongst the groups, F (2, 78) = 1.15, p = .324.

Lastly, we explored differences in aspects of social cognition, particularly emotional intelligence and empathy. Students also did not differ in their reported empathic responses on the Toronto Empathy Questionnaire (Spreng et al., 2009), F (2, 78) = .18, p = .834, but as noted elsewhere this measure does not distinguish cognitive

from emotional components of empathy (Baker & Good, 2012). There was no significant difference amongst injury severity groups on the composite score on the clinical measure of emotional intelligence (EQ-i), F (2, 78) = .01, p = .986, nor the research-based measure (EI; Barchard, 2001), F (2, 78) = 1.25, p = .293.

Lastly, we hypothesized that although persons with head injury and moderate/severe TBI groups may differ in emotional intelligence (EI; Barchard, 2001), we expected history of head trauma to account for unique variance in affect recognition scores (ACS-Affect Recognition) over and above emotional intelligence and psychiatric health (i.e., SA-45). Regression analyses demonstrated that severity of head trauma (noMHI, MHI, moderate/severe TBI) significantly predicted the ability to recognize emotional expressions (ACS-Affect Recognition) over and above other indices of emotional functioning (emotional intelligence, psychiatric health [SA-45 Global Severity Index]), F (1, 78) = 5.19, p = .026, accounting for 6.3% of the variance[16].

Discussion

Across a variety of measures we have provided evidence of a pattern of emotional underarousal for persons who have sustained either mild head trauma or moderate/severe TBI relative to their peers who report no history of head injury. We demonstrated this emotional underarousal in terms of autonomic indices of significantly attenuated resting EDA amplitude, resting heart rate, as well as the pattern of a blunted CAR for persons with MHI or moderate/severe TBI relative to persons who have not sustained injury to the head/brain. The former findings mirror our results in other studies (e.g., Baker & Good, 2010; 2012; 2014; Jung & Good, 2007) and are similar to literature of persons

[16] Portions of this data will be presented at the 91st Annual Conference of the American Congress of Rehabilitation Medicine, October 2014.

with moderate/severe disruption to the VMPFC in terms of reduced physiological responsivity (e.g., Damasio et al., 1990; Hopkins et al., 2002). Although students across all three groups responded to the emotionally-evocative stimuli in terms of increased self-reported arousal, EDA, and heart rate, we did not evidence differential responding across the testing session as a function of severity of injury, although a main effect of reduced arousal was demonstrated for the head injury and TBI groups. Notably, mean salivary cortisol responsivity to the emotional arousal manipulation was not as hypothesized. The average levels of salivary cortisol decreased across the testing session for all groups regardless of injury severity. The peak cortisol response to stressors such as the Trier Social Stress Test is typically observed approximately 30 minutes from the onset of the stressor stressor and the recovery to a baseline measure of cortisol often occurs within an hour (see Dickerson & Kemeny, 2004; Kirschbaum et al., 1992; Kudielka, Wust, et al., 2007). However, we did not observe any increases in salivary cortisol across the testing session for any of the groups. Although the emotionally-evocative stimuli produced sufficient increases in autonomic indices of arousal, it is possible that we did not see an increase in arousal as indexed by salivary cortisol levels in the current study because this task lacked a social evaluative component and/or a performance demand. These latter two components (e.g., such as the Trier Social Stress Test [TSST] – for discussion see Dickerson & Kemeny, 2004) have been suggested to produce more robust, consistent increases in salivary cortisol.

Based on our prior research and the Yerkes-Dodson law (1908), we hypothesized that cognition would be advantaged by increasing arousal for persons with a history of mild head trauma and moderate/severe TBI, whereas performance would be poorer for

persons with no history of head trauma after exposure to the emotionally-evocative stimuli. Even though the students' emotional arousal increased in response to the arousal manipulation in terms of autonomic responses and self-reported arousal (but not for neuroendocrine measures), we did not find strong support for this hypothesis. Dickerson and Kemeny (2004) have discussed that salivary cortisol responses to stress paradigms may have varying and oftentimes conflicting results, although it is unclear why the effects are so varied (e.g., Kudielka & Wust, 2010; Kudielka, Hellhammer, & Wust, 2009). Furthermore, Hellhammer, Wust, and Kudielka (2009) mentioned that self-reported measures of arousal may not reflect the pattern of endocrine responsivity as we have also shown in the current study. It is likely there may be differential effects on cognition as a function of sympathetic versus endocrine reactivity (e.g., see Lupien et al., 2007). Unlike epinephrine which exerts indirect effects on the brain via vagus nerve activity, glucocorticoids can traverse the blood brain barrier and act directly on receptors (Lupien et al., 2007). Stress hormones affect cognition by binding to specialized receptors in regions that have been identified to be associated with such abilities (e.g., hippocampal and PFC regions - Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009; Jaferi & Bhatnagar, 2007; Lupien et al., 2007; McEwen & Sapolsky, 1995). Therefore, it is possible that because salivary cortisol concentrations did not increase in response to the emotional arousal induction we did not consistently observe the expected effects on cognitive abilities that are associated with PFC and hippocampal function (e.g., visuospatial memory, attention). In light of the glucocorticoid receptor ratio hypothesis by de Kloet and colleagues (de Kloet et al., 2008; de Kloet, 2000) it is possible that cortisol levels were too low to sufficiently activate Type II receptors (which are

preferentially located in the PFC and hypothalamic regions – see Jaferi & Bhatnagar, 2007; Lupien & McEwen, 1997; Nicolson, 2008) to observe any behavioural effects. Furthermore, it is possible that even though the emotional arousal task in this study was an abbreviated version from another study that was shown to be effective in modulating arousal state (Baker & Good, 2012), it is possible that the revised task in the current study was too short in duration to produce the hypothesized effects. Although we attempted to mimic exposure to daily stressors (e.g., watching emotionally arousing media) to increase arousal, static images may not reflect the magnitude of real-life experiences.

Two other key findings were evidenced in this study. First, we found support for the hypothesis of dysregulated stress responsivity in terms of preparation to respond to challenges throughout the day (i.e., the CAR; Clow et al., 2010; Pruessner et al., 1997**).** To our knowledge this is the first study to demonstrate that persons with moderate/severe TBI, as well as persons with 'mild' head injury, do not produce the typical elevation in cortisol concentrations after morning awakening (see Clow et al., 2010; Pruessner et al., 1997) whereas persons with no history of head trauma produce a burst of cortisol upon awakening (see Figure 3.11). This striking finding provides novel insight into emotional dysregulation for persons with trauma to the head/brain in terms of dysregulated stress response for preparation to respond to daily stressors. This finding demonstrates that the assessment of cortisol responses to awakening in the TBI population may serve as a useful tool in identifying even subtle changes in HPA functioning. We suggest that blunted/atypical CAR evidenced in this study is related to the effects of injury to the brain which are not, based on this evidence, transient, and suggest dysregulation of the HPA axis (e.g., Clow et al., 2010). We also suggest that the CAR may serve as a new

biomarker of residual emotional challenges postinjury which warrants further examination and replication. The CAR has also been shown to be atypical in other populations (e.g., posttraumatic stress disorder – see metaanalysis by Chida & Steptoe, 2009). Similarly, other indices of hormonal dysfunction and deficiencies after TBI such as abnormal thyroid functioning or anterior hypopituitarism have shown deficient gonadotrophin, thyroid stimulating hormone, and growth hormone levels. Of particular interest, a review by Benvenga, Campenni, Ruggeri, and Trimarchi demonstrated that 53% of 367 case reports of persons with posttraumatic hypopituitarism following closed head injury (94% of sample) had deficient adrenocorticotrophin hormone (ACTH) levels. ACTH is secreted by the anterior pituitary in response to corticotrophin-releasing hormone (CRH) from the hypothalamus and is involved in the stimulation of the release of glucocorticoids i.e., cortisol (see McCormick, 2007, for a review). ACTH is involved in the cascade of hormonal events during a stress response. Therefore, given the atypical ACTH levels reported by others (Benvenga et al., 2000) it is possible that persons with trauma to the head/brain that demonstrate atypical cortisol responses (i.e., a blunted CAR) may also have disrupted levels of anterior pituitary hormones. It is possible that persons in our sample may also evidence a pattern of hypopituitarism because of the vulnerability of the pituitary to injury in TBI (see Agha & Thompson, 2006). The blunted CAR suggests atypical hormonal function that may be a peripheral indicator of dysregulated stress system function. Therefore, examination of other measures of pituitary functioning (e.g., ATCH levels, GH, and others) is warranted in this population especially when persons have past the acute phase of recovery because this research is very limited (see Agha & Thompson, 2006; Benvenga et al., 2000). The cognitive,

emotional, physical, and social sequelae have been the primary emphasis of the literature on TBI, but it has been proposed that persons with TBI may present with hypopituitarism that is often overlooked and may account for some of the neurobehavioural sequale postinjury (Agha & Thompson, 2006).

Secondly, although we did not find significant main effects of injury severity with measures of social cognition (i.e., emotional intelligence, empathy - but see Baker & Good, 2012; Leopold et al., 2012), we provided evidence that severity of injury significantly predicted the ability to identify emotional facial expressions over and above indices of emotional functioning (i.e., psychiatric health SA-45 Global Severity Index) and emotional intelligence (EI, Barchard, 2001). In general, an overall poorer ability to label facial expressions of emotions (e.g., sad, surprised, angry, surprised, disgusted, neutral expression) was related to increased injury severity. This finding is in line with the body of research that has demonstrated significant impairments in ability to label/identify emotional stimuli following TBI (e.g., Bornhofen & McDonald, 2008; Milders, Fuchs, & Crawford, 2003; Spikman et al., 2013; Williams & Wood, 2010). For example, Williams and Wood (2010) found that persons with moderate/severe TBI were significantly impaired at recognizing emotional facial expressions, especially negative emotional expressions, relative to a control group. In another study, Borago, Prigatano, Kwasnica and Rexer (2003) demonstrated that persons with ‘complicated’ and ‘uncomplicated’ mild TBI performed worse on affective measures relative to persons with no history of TBI. Furthermore, Borago et al. demonstrated with discriminant function analysis that affective disturbance (measured via Affect subtest on the BNI Screen for Higher Cognitive Functions) was best able to discriminate between TBI and

control groups in this study with 80% and 90% of cases correctly classified, respectively. We, too, have demonstrated and highlight the affective disturbances following mild or moderate/severe TBI.

Limitations. Although salivary cortisol samples were taken across two days, it is important to mention that we only measured the CAR on a single day. As such, repeated assessment of the CAR across multiple days is important to examine for consistency of response. Furthermore, a variety of variables may moderate or mediate the relationship of blunted cortisol responsivity in terms of the CAR that have yet to be included in a more comprehensive model and/or have not been thoroughly studied to date.
However, we examined the potential influence of variables such as level of self-reported depressive symptoms and morningness/eveningness with respect to the CAR and did not find the results to vary (e.g., used as a predictor in an hierarchical regression; Analysis of Covariance). Furthermore, we excluded participants from this study if they endorsed factors that may be related to altered salivary cortisol such as shift work, use of glucorticoids and/or other medications.

Another limitation of the study is that we did not obtain medical records for persons with mild head trauma and relied on self-report method because the majority (i.e., 60%) of the persons in our study did not seek medical attention regarding the head trauma event. Although this method of ascertainment of a history of head trauma may be fraught with challenges (e.g., accuracy or bias of reporting – see Harrison, McLaughlin, & Coalter, 1996), it is not unlike the information obtained via clinical interview. Furthermore, persons with self-reported head injury are often overlooked in epidemiological studies because persons with such injuries often do not seek medical

attention (e.g., Sosin, Sniezek, & Thurman, 1996); however, considering that persons who do not seek treatment for the head trauma may have sustained relatively subtle injury (as compared to those with emergency department admission), our findings are even more striking because these persons are likely at the very 'mild' end of the spectrum of injury severity (Alexander, 1995; Iverson & Lange, 2009). We also acknowledge that the generalizability of this study may be limited in that our study was conducted with university students.

Conclusions. Our findings highlight the importance of emotional functioning postinjury and the potential dysregulation of emotional arousal postinjury, especially the CAR and attenuated indices of physiological arousal at rest (e.g., EDA, heart rate). Further examination of the CAR as an indicator of emotional dysregulation is warranted for persons with TBI. As well, evidence of affective challenges in this study such as an index of emotional intelligence predicting ability to recognize others' emotional facial expressions require replication, but provides preliminary evidence of altered aspects of social cognition for persons with a history of head trauma.

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General Discussion

TBI is one of the most concerning health problems in the world and is a leading cause of death and disability (WHO – Neurological Disorders Report, 2006; Teasell, Aubut, Bayley, & Cullen, 2013 – EBR – ABI). The complexity of TBI and its sequelae are not homogeneous (McAllister, 2008; 2011); however, the literature consistently demonstrates changes in emotional functioning, cognition, and behavioural outcomes after TBI which may impede successful reintegration (Ponsford, 2013; Zasler, Katz, & Zafonte, 2007). Although the duration of sequelae postinjury for persons with 'mild' injury has often been debated (Carr, 2007; Chuah, Mayberry, & Fox, 2004; Dikmen, McLean, & Temkin, 1986; Len & Neary, 2011), there are data to suggest impairments in cognitive abilities (e.g., Raskin, Mateer, & Tweeten, 1998; Chuah et al., 2004), affective domains (e.g., Borgaro, Prigatano, Kwasnica, & Rexer, 2003; Panayiotou, Jackson, & Crowe, 2010), the experience of postconcussive symptomology (Dean, O'Neill, & Sterr, 2012), and physiological arousal are not transient (Baker & Good, 2014). Here, across several studies we have provided evidence of alterations in emotional and social functioning as a function of a history of head trauma with a neurobehavioural profile that follows a continuum of injury severity (mild, moderate/severe – Alexander, 1995; Iverson & Lange, 2009). Overall, persons with a history of head trauma present with a profile consistent with dampened emotional arousal and emotional functioning relative to persons who have not experienced head trauma.

The studies that comprise this dissertation research are unique in several respects. First, as with other studies conducted in our lab (e.g., Baker & Good, 2014; van Noordt & Good, 2011) we did not advertise interest in a history of head trauma to avoid any

possible 'diagnosis threat' or biases (e.g., Suhr & Gunstad, 2002) – participants were naive to the purpose of the studies until they were debriefed. Secondly, in each study the experimenters were blind to the head injury status of the participant until the end of the testing session to avoid experimenter bias[17]. Thirdly, information regarding the head injury event was embedded within multiple health-related questions so it is unlikely that participants were aware that this was a variable of interest during their participation in the research until debriefed. Given this approach it is unlikely that bias/expectations of performance as a result of prior head trauma influenced the behavioural and self-reported profiles observed in these studies.

More importantly, this dissertation uniquely examined emotional and social functioning in terms of both self-reported and physiological indices with persons with 'mild' head injury and across the spectrum of injury severity. We primarily tested the model of emotional volatility/lability or hyporesponsiveness (i.e., emotional underarousal) and its implications for social behaviours as well as the potential impact of modifying arousal and its relation to cognitive performance. The examination of components of social cognition (i.e., emotional intelligence and empathy; Adolphs, 2001) and their potential relationship to emotional and social functioning is also relatively unique with this population especially for those with mild head trauma (although see de Sousa et al., 2010; 2011; 2012; Spikman, Timmerman, Milders, Veenstra, & Van der Naalt, 2013 for examination with moderate/severe TBI). The third study is especially novel with respect to the exploration of neuroendocrine indices of stress responsivity, particularly the cortisol awakening response (CAR; Pruessner et al., 1997; Clow,

[17] Except for Baker and Good (2014b) which included persons with moderate/severe TBI which, at times, the behaviour of the participant may have indicated more substantial injury.

Hucklebridge, Stalder, Evans, & Thorn, 2010) and salivary cortisol responsivity to an experimental emotional arousal induction for persons who have experienced trauma to the head/brain.

In the first study, *Emotional and Social Functioning of University Students with and without Mild Head Injury* we examined the self-reported emotional and physical health as well as social functioning in university students. Postconcussive symptom reports were of particular interest in this study. A fundamental issue exists in the literature regarding the etiology and duration of postconcussive symptom experiences as a function of sustaining mild TBI as well as the nonspecificity of symptoms (e.g., see Carr, 2007; King, 2003; Mittenberg & Strauman, 2000). Postconcussive-like symptoms have been commonly reported by the university student population (Gouvier et al., 1992; Iverson & Lange, 2003). Therefore, we examined the postconcussive symptom report profiles of students with and without a history of mild head injury (MHI) in this study. We measured self-reports of postconcussive symptoms via the Postconcussive Syndrome Checklist (PCSC; Gouvier et al., 1992) as well as indices of emotional and social functioning in 230 university students who were naïve to the purpose of the study.

We expected (as in Baker & Good, 2010) that students with a self-reported prior head trauma would experience postconcussive symptoms with more frequently, for longer durations, and with greater intensity than students without a history of head injury. We found support for this hypothesis in that students with self-reported MHI (N = 98; 42% of sample) endorsed postconcussive symptoms significantly more often, experienced them for longer durations, and with greater intensity than their peers who did not report any head injury. We compared the average PCSC scores in this study to other

studies of university students (e.g., Sawchyn, Brulot, & Strauss, 1999, etc. see Table 1.2) and found the baseline symptom reports to be similar; however, our study demonstrated magnified experiences of these symptoms for students with MHI relative to students without head trauma. In particular, significant differences were found between students with and without MHI for reports of dizziness, memory problems, and concentration difficulties. Moreover, it has been reported that stress may excacerbate symptom reporting (e.g., Gouvier et al., 1992); therefore, we examined the role of stressful life events with postconcussive symptom reporting as a function of a history of mild head trauma. We found that, overall, students with a self-reported MHI acknowledged significantly more experiential stress relative to their peers. We also found (as in Gouvier et al., 1992) that postconcussive symptom reporting was significantly related to increased life stressors for both students with and without MHI. Yet, when we controlled for the influence of experiential stress on postconcussive symptom reports (via ANCOVA) we found a trend for an effect of MHI history in that those with MHI still reported experiencing postconcussive symptoms more so than their peers.

It has been suggested that preinjury characteristics may play a role in increased symptom reporting such as income, level of education, psychiatric distress, and time elapsed since injury. We suggest, as do others (e.g., Gordon, Haddad, Brown, Hibbard, & Sliwinski, 2000), that the increased postconcussive symptom reported by persons with TBI relative to persons without prior head trauma are primarily related to the injury and not to other factors. For example, Gordon et al. (2000) examined postconcussive symptom reporting across 6 groups: mild TBI (n = 135), moderate/severe TBI (n =275), persons with no disability (n = 287), those with spinal cord injury (n = 107), persons with

HIV positive (*n* = 197), and those who had a liver transplant (*n* = 107). Overall, Gordon et al. found that persons with either mild TBI or moderate/severe TBI reported significantly more symptoms than the other groups. Their finding demonstrates that regardless of injury severity postconcussive symptom reports may be an indicator of the sequelae postinjury. Further, Gordon et al.did not find preinjury characteristics (as reported by a family member) such as age, sex, level of education, income, psychiatric health (i.e., depression) and ethnicity, nor time elapsed since injury, to be significant predictors of symptom reports for persons with TBI (mild, moderate/severe).

On another note, it has been suggested that difficulties in social interactions postinjury for persons with moderate/severe TBI may be a result of impaired emotional functioning and/or aspects of social cognition (Bornhofen & McDonald, 2008; Ganesalingam, Sanson, Anderson, & Yeates 2006; Yeates et al., 2004); therefore, a second major goal of the first study was to examine indices of emotional functioning (Emotional Intelligence [EI] – Barchard, 2001; Callous Affect subscale of SRP-III, Paulhaus et al., in press) and their potential relationship to social behaviours (Antisocial Behaviour subscale of SRP-III, Paulhaus et al., in press). Overall, students with self-reported MHI endorsed socially unacceptable behaviours and an erratic lifestyle significantly more often than students with no history of MHI. Furthermore, we observed a trend of attenuated emotionality in terms of higher scores on the Callous Affect subscale of the SRP-III for students with self-reported MHI when compared to students with no head trauma. This dimension of callous affect is characteristic of generally deficient emotional sensitivity (e.g., Hare & Neumann, 2008) and is similar to the underarousal/dampened emotional responsivity that is seen in persons with disruption to

the VMPFC region (e.g., Damasio, Tranel, & Damasio, 1990; 1998; Hopkins, Dywan, & Segalowitz, 2002). We did not find significant differences in the measure of emotional intelligence (EI; Barchard, 2001), however. Notably, although males scored higher than females on the SRP-III measures, a regression analysis demonstrated that MHI significantly accounted for unique variance over and above sex for socially unacceptable behaviours. Lastly, we demonstrated that total scores on the emotional intelligence measure significantly predicted socially unacceptable behaviours, but the model was significant only for students who reported a MHI and accounted for approximately 20% of the variance in socially unacceptable behaviours (SRP-III Antisocial Behaviour subscale) such that lower scores on the EI questionnaire predicted poorer social functioning. This finding suggests that a component of social cognition, emotional intelligence, predicts social interactions for persons with mild head injury. Similar findings have been shown across the spectrum of injury severity i.e., with persons with moderate/severe TBI (e.g., see deSousa et al., 2011; 2012; Leopold et al., 2011 for similarities). It it possible that persons with mild head injury, like those with moderate/severe TBI (e.g., Riggio, 2011), also experience challenges in social interactions.

In the second study, *Emotional Functioning and Reactivity of University Students as a function of a History of Mild Head Injury* we further examined emotional underarousal in terms of responsivity to emotionally-evocative stimuli. We explored changes in emotional arousal in terms of autonomic (e.g., EDA) and self-reported indices. We also examined if the affective experience would effectively modify arousal for persons with MHI for a substantive period of time. We expected that persons with MHI

would demonstrate reduced responding overall (i.e., underarousal), especially to stimuli with negative valence, in a fashion similar to persons with moderate/severe TBI (Hopkins et al., 2002) and or lesions to the VMPFC (Damasio et al., 1990). We replicated our previous findings of underarousal (Baker & Good, 2014) in that students with self-reported MHI (34.9%; *N* = 30) produced significantly attenuated physiological arousal in terms of EDA amplitude that followed a gradient of injury severity (noMHI, MHI with altered state of consciousness, MHI with loss of consciousness) despite reporting a pattern of experiencing more life stressors. We also found evidence (trend) of reduced emotional reactivity in that students with MHI reported decreased responsivity to others' distress (EI subscale) and reported more positive emotionality. The former pattern suggests that persons with MHI may have challenges in emotional 'empathy'. This finding may be consistent with a positivity bias ('rose-coloured view' – Beer et al., 2003; 2006; 2010; Beer & Hughes, 2010; Beer, Lombardo, & Bhanji, 2010) for persons with MHI (often referred to as 'la belle indifference' for persons with moderate/severe TBI [Janet] 1849-1947 in Iezzi, Duckworth, & Adams).

Furthermore, students' EDA and self-reported arousal increased as a result of the experimental manipulation of arousal (i.e., exposure to the emotionally-evocative stimuli [positive/pleasant, negative/unpleasant, and neutral/ambiguous from the International Affective Picture System [Lang, Bradley, & Cuthbert, 2008) and maintained the increase in physiological arousal for at least 30 minutes. This manipulation was more effective at modifying arousal state for persons with MHI than a previous manipulation of a psychosocial stressor (Baker & Good, 2014). Even though we did not find support for our hypothesis that the valence ratings of the emotionally-evocative stimuli would differ

between students with and without MHI, we found striking evidence of reduced and/or indiscriminate emotional responding to the stimuli for students with a history of MHI. For students with MHI, EDA amplitude responses were similar to negative, positive, and neutral/ambiguous stimuli, whereas the students with no history of head injury produced larger EDA responses to negative, positive, and ambiguous stimuli (higher responses for negative pictures). This finding demonstrates reduced responsivity and/or indifference to affective stimuli and mimics, albeit subtly, that observed by Hopkins et al. (2002) and others (de Sousa et al., 2011; 2012) for persons with moderate/severe TBI. We suggest that the attenuated responsivity and indiscriminatory response to the emotionally-evocative stimuli for persons with mild head trauma is related to the reduced emotional capacity observed following TBI in general (Damasio et al., 1990).

In the third study, *Neuroendocrine and Autonomic Indices of Stress Responsivity for Persons with Traumatic Brain Injury and Mild Head Injury* we replicated our findings of emotional underarousal (as in Baker & Good, 2014), but also uniquely explored neuroendocrine aspects (i.e., salivary cortisol; cortisol awakening response [CAR]) and autonomic indices (i.e., EDA) of emotional dysregulation in terms of stress responsivity across the spectrum of injury severity (students with MHI [n = 32]; students with moderate/severe TBI [n = 9], and age and education matched controls [n = 40]). We found striking evidence of emotional underarousal in terms of resting EDA and heart rate for head/brain injury groups relative to students with no history of head injury. Although students responded to the emotional arousal manipulation (as they did in Baker & Good, 2012 [Study 2]), we did not find consistent support for our hypothesis that cognitive performance would be advantaged via exposure to these emotionally-evocative stimuli

for persons with head/brain injury and would be disadvantaged for persons with no history of head injury based on the the Yerkes Dodson law (see Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; also see de Kloet et al., 1997; de Kloet, 2000). The emotional arousal manipulation did not induce sufficient increases in salivary cortisol levels for any of the groups so it is possible that this may account for the limited evidence of cross-over interactions on measures of cognitive ability as a function of a history of head/brain injury (see de Kloet et al., 1997).

There were two other major findings from this study that will contribute substantially to the literature and our understanding of emotional functioning and dysregulation following TBI. First, although we did not find evidence of main effects of injury severity for social cognition measures (i.e., emotional intelligence or empathy), we demonstrated that severity of injury significantly predicted performance on an affect recognition task (ACS- Affect Recognition; Wechsler, 2009) over and above other indices of emotional functioning (i.e., Global Severity Index of the SA-45; Strategic Advantage, 2008). This finding is in concert with the current literature regarding impairments in the ability to label/identify emotional facial expressions following moderate/severe TBI (e.g., Bornhofen & McDonald, 2008; Milders et al., 2003; Spikman et al., 2013; Williams & Wood, 2010). This finding also demonstrates that emotional challenges postinjury follow a gradient of injury severity. The ability to identify others' emotional expressions has important implications for social functioning such as being able to provide appropriate/expected responses to others based on their emotional state. It is plausible that the socioemotional difficulties persons with MHI and TBI encounter postinjury are a result of the decreased ability to detect/identify socioemotional cues (i.e.,

facial expressions) in their environment. Furthermore, their reduced physiological response to emotionally-charged stimuli may contribute to this challenge in that persons with head trauma produced significantly attenuated EDA to all stimulus types (positive, negative, and ambiguous) relative to those with no history of head trauma and that they did not produce differential responses to the stimulus types (i.e., was not heightened to stimuli of negative valence). Similarly, it is possible that this emotional underarousal plays a role in their ability to respond to other stimuli such as facial expressions of emotion.

Secondly, we demonstrated that students with moderate/severe TBI *and* students with MHI did not produce the typical cortisol awakening response (see Clow et al., 2010) and demonstrated blunted change, if any, in salivary cortisol levels within the first 45 minutes of wakening, whereas students with no history of head trauma produced the typical CAR showing increased salivary cortisol upon awakening in preparation to respond to daily challenges (Clow et al., 2010; Pruesnner et al., 1997). As suggested by others such as Clow and colleagues (2010), CAR is a promising biomarker of emotional and psychosocial health, and the findings from our study demonstrate that it may be a potentially useful marker of emotional/stress dysregulation in the TBI population regardless of injury severity. Furthermore, this finding may also suggest anterior pitiuitary dysfunction many years postinjury (i.e., posttraumatic hypopituitarism – see Agha & Thompson, 2006; Benvenga, Campenni, Ruggeri, & Trimarchi, 2000). To our knowledge this is the only study to examine the CAR in the TBI population. Replication of this study is necessary, with repeated daily measures of the CAR over a few days, but the data supports the underarousal hypothesis quite well and implicates hypothalamic-

adrenal-pituitary axis disruption in TBI, and MHI, in general. Future research should examine other indices of emotional disruption via physiological indices such as salivary alpha amylase (Nater & Rohleder, 2009) or adrenocorticotrophin hormone (ACTH) to extend these findings.

The indicators of emotional underarousal observed in this research is in concert with the theoretical framework of the *somatic marker hypothesis* (i.e., Damasio et al., 1990). The main theme of this hypothesis is that the decision making process is influenced by somatic markers/signals. Their theory suggests that emotions may be described as representations of physiological responses referred to as somatic markers. For example, Bechara, Damasio, Damasio, and Anderson (1994) have shown that persons with damage to the ventromedial prefrontal cortex (VMPFC) have profound impairments in decision making, which they suggest is likely due to their unresponsiveness to the future consequences of their advantageous or disadvantageous decisions. Damasio and colleagues (e.g., 1998) have suggested that the interpretation of the [emotional] somatic markers (i.e., bodily signals) that bias decision making is impaired for persons with disruption to the VMPFC. As previously noted, the VMPFC is involved in the modulation of emotional responding (see Naqvi, Shiv, & Bechara, 2004; Wallis, 2007), so it is not surprising that Damasio and colleagues have also demonstrated that persons with damage to the VMPFC demonstrate flattened EDA responses during decision making tasks (e.g., see Tranel, 2000 for review). We (e.g., Baker & Good, 2012; 2014a) suggest that persons with mild head trauma present with a flattened behavioural profile that is similar to that of persons with moderate/severe injury to the VMPFC. We propose that it is possible that not only is the interpretation of the somatic markers challenged

after TBI, but also that the production of these signals may be attenuated postinjury based on our findings of emotional underarousal. Furthermore, based on our findings (e.g., Baker & Good, 2012), we suggest that the indiscriminate responses and/or insensitivity to stimuli are a function of emotional underarousal that is evident with persons with even mild head trauma. There is little, to no, influence of affective stimuli on physiological arousal for persons with head trauma. It has been suggested that the somatic signals produced warn and/or alert the individual to whether the stimuli is 'negative' or 'positive' (see Bechara, Damasio, & Damasio, 2000). Based on our data it is possible that persons with mild head trauma do not produce these somatic signals to the same degree as persons without a history head trama.

Limitations

A few limitations of these studies must be mentioned. A major limitation of the current research is that we lacked corroborating medical and/or neuroimaging evidence to demonstrate anatomical, structural, and/or network disruption for students with self-reported head trauma (e.g., white matter integrity in DTI – see Belanger, Vanderploeg, Curtiss, & Warden, 2007). However, as previously mentioned the majority of persons who sustain 'mild' head trauma do not seek medical attention and at least half of the students in our samples reported not seeking treatment for their injury – therefore medical documentation was unattainable (e.g., Sosin, Sniezek, & Thurman, 1996). Even if we did have medical documentation such as computerized tomography (CT), relatively few persons with mild head trauma who seek medical treatment demonstrate structural abnormalities on CT (e.g., only 13% had positive CT findings in Bouida et al., 2013) and CT does not capture the metabolic disruption (Bigler, 2013; Giza & Hovda, 2001). Even

though this fact may viewed as a limitation, this research is important because oftentimes persons with undocumented, but substantive, mild head injury are overlooked in both epidemiological studies and in experimental research. Furthermore, our findings are even more striking as it is likely that those who do not seek medical treatment may have sustained more 'minor' injuries and are at the very mild end of the spectrum of injury severity (Alexander, 1995; Iverson & Lange, 2009).

Another limitation of these studies is that measures of behaviours were via self-report and self-reflection may be prone to issues with this population (e.g., anosoagnosia). However, the questionnaires were primarily objective and, otherwise, standardized. Further, for many tests, the statements that were responded to consisted with concrete descriptions of behaviour (e.g., I never cry at movies; I have never been arrested), a problem not typically associated with the anosagnosia – that is, the person will often acknowledge a particular behaviour, but not appreciate its impact on self or others (i.e., lacks the insight or reflective nature of a behaviour). Future research should obtain collateral information from friends, family, and co-workers regarding social and emotional functioning because it is possible that persons with MHI, as with those with moderate/severe TBI, in order to examine discrepencies, or compare, insights into their overall functioning including socially unacceptable behaviours (e.g., Lezak, Howieson, Bigler, & Tranel, 2012). Physiological indices of emotional functioning illustrated a pattern of hyporesponsiveness, in general, which is in line with the current literature on persons with moderate/severe TBI (e.g., Leopold et al., 2011; de Sousa et al., 2011; 2012; Bornhofen & McDonald, 2008; McDonald, 2013).

Lastly, we acknowledge that the generalizability of this study may be limited in that our study was conducted with university students. Persons who sustain head trauma and then proceed to attend university may be a selective group in that they may have a better preinjury intellectual capacity, or milder injuries and concussions pre-university, than their cohort because following the injury they pursue post-secondary education. Furthermore, it is possible that those that sustain a head trauma and pursue post-secondary education are better able to cope with the demands of university life than their head injury cohort that do not attend university. It is also important to note that it is possible that this group may consist of persons who are not as emotionally labile and/or overreactive as other persons who have sustained a head trauma and are not attending university. Therefore, it is possible that our head injury sample consists of persons who are hypoaroused and therefore presented with emotional underarousal in terms of physiological measures. In this case, our findings may not be replicated in a general head trauma population in the community which may include both those who are hyperaroused and those who are hypoaroused.

Conclusions

In conclusion, this research is unique in that socioemotional functioning in terms of indices of stress responses (self-report or physiological) and emotional intelligence are rarely examined in the TBI population. Furthermore, to our knowledge, this trajectory of research is unique with respect to the emphasis on the continuum of the severity of brain injury (Alexander, 1995; Iverson & Lange, 2009). Across these studies we have provided consistent evidence of changes in emotional and social functioning for persons with mild and moderate/severe injury even many years postinjury. Although not all comparisons

were significantly different between head injury groups, the pattern of reduced emotional responsivity, poorer emotional recognition ability, and social behaviours were illustrated to follow a gradient of severity of injury. The emotional and social changes observed in the 'mild' injury population mirror that, albeit subtly, of persons with moderate to severe TBI. We have provided striking evidence of emotional underarousal in this population and also have identified potentially useful indices of dysregulated stress responsivity (i.e., cortisol awakening response) for both persons with mild head injury and those with moderate/severe TBI. This dissertation research primarily illustrates that the spectrum of injury severity of TBI follows a continuum (Alexander, 1995; Iverson & Lange, 2009) and that the neurobehavioural sequelae are quite similar across the injury severity continuum, albeit subtly presented for persons with self-reported 'mild' head injury. We, too, (e.g., Leopold et al., 2011; Bornhofen & McDonald, 2008) suggest that emotional functioning should be considered a primary variable for problems that interfere with social integration, even for persons with mild head injury. Continuation of such research will propel further insight into the complexity of brain-behaviour relationships and perhaps provide a better understanding of the functioning of persons who have sustained brain injury.

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APPENDIX A

Subject: REB 08-236 - Accepted as Clarified
From: Research Ethics Board <reb@brocku.ca>
Date: 03/03/2009 9:24 AM
To: "'dgood@brocku.ca'" <dgood@brocku.ca>, "'js01cb@brocku.ca'" <js01cb@badger.ac.brocku.ca>, "'ad03cr@brocku.ca'" <ad03cr@badger.ac.brocku.ca>
CC: "'Michelle McGinn'" <rebchair@brocku.ca>

DATE: March 3, 2009

FROM: Dan Malleck, Acting Chair
Research Ethics Board (REB)

TO: Dawn GOOD, Psychology
Julie St. Cyr-Baker, Angela Dzyundzyak

FILE: 08-236 GOOD
Faculty Research

TITLE: Individual Differences in Personality and Health

The Brock University Research Ethics Board has reviewed the above research proposal.

DECISION: ACCEPTED AS CLARIFIED

This project has received ethics clearance for the period of **March 3, 2009 to February 28, 2010** subject to full REB ratification at the Research Ethics Board's next scheduled meeting. The clearance period may be extended upon request. ***The study may now proceed.***

Please note that the Research Ethics Board (REB) requires that you adhere to the protocol as last reviewed and cleared by the REB. During the course of research no deviations from, or changes to, the protocol, recruitment, or consent form may be initiated without prior written clearance from the REB. The Board must provide clearance for any modifications before they can be implemented. If you wish to modify your research project, please refer to http://www.brocku.ca/researchservices/forms to complete the appropriate form Revision or Modification to an Ongoing Application.

Adverse or unexpected events must be reported to the REB as soon as possible with an indication of how these events affect, in the view of the Principal Investigator, the safety of the participants and the continuation of the protocol.

If research participants are in the care of a health facility, at a school, or other institution or community organization, it is the responsibility of the Principal Investigator to ensure that the ethical guidelines and clearance of those facilities or institutions are obtained and filed with the REB prior to the initiation of any research protocols.

The Tri-Council Policy Statement requires that ongoing research be monitored. A Final Report is required for all projects upon completion of the project. Researchers with projects lasting more than one year are required to submit a Continuing Review Report annually. The Office of Research Services will contact you when this form *Continuing Review/Final Report* is required.

Please quote your REB file number on all future correspondence.

DM/an

REB 08-236 - Accepted as Clarified

Research Ethics Office
Brock University
Office of Research Services, MC D250A
500 Glenridge Avenue, St. Catharines, ON L2S 3A1
Phone 905-688-5550 ext. 3035
Fax 905-688-0748
Email: reb@brocku.ca
http://www.brocku.ca/researchservices/Ethics_Safety/Humans/Index.php

APPENDIX B

Participants Needed!!!

For research investigating

INDIVIDUAL DIFFERENCES IN PERSONALITY AND HEALTH

As a participant you will be asked to complete a variety of questionnaires and will be eligible for **~ 1.5 research participation hours**.

To participate in this study you must be fluent in English.
For more information or to participate please contact

Julie St. Cyr-Baker
(js01cb@brocku.ca)

Angela Dzyundzyak
(ad03cr@brocku.ca)

Supervisor: Dr. Dawn Good Dawn.Good@brocku.ca (ext. 3556)

This study has been reviewed by and received ethics clearance through the Office of Research Ethics, Brock University (REB 08-236) 905-688-5550 ext. 3035

INDIVIDUAL DIFFERENCES IN PERSONALITY AND HEALTH

Dear Participant*:*

You are invited to participate in a survey we are conducting to examine individual differences in personality and health. This research is facilitated by Dr. Dawn Good, Julie St. Cyr-Baker, and Angela Dzyundzyak in the Psychology Department at Brock University.

Participation in this study is entirely voluntary and involves completing a questionnaire package which is expected to take about ***one and a half hours*** of your time. The questions are quite varied with respect to personality (e.g. do you enjoy driving fast?) and health (e.g. have you ever been hospitalized for a serious illness?). Some of the questions are personal and sensitive in nature. You will also be asked to provide background information about yourself such as sex, age, and level of education.

You may omit any question you prefer not to answer, and you may withdraw from the study at any time without consequences to yourself. Should you choose to withdraw from the study at any point in time your data will be destroyed and not included in any analyses. All information you provide will be considered confidential. Your data will be grouped with other participants' data and any information gathered from this study used in discussions, publishable articles, or presentations will be summarized and refer only to group results, preserving anonymity.

The data collected through this study will be kept in a secure location to which only the researchers and research assistants who have completed confidentiality agreements will have access. The data will be kept for a period of 5 years after which time it will be destroyed. There are no known or anticipated risks for participation in this study.

By volunteering for this study, you will learn about research in psychology in general and the topic of this study in particular due to your first-hand experience. The information from this study will contribute to research on individual differences in personality and health. In addition, you will receive a detailed debriefing form about the study. You will be invited to view the results of this study at its completion (by February 1, 2010). Also, you may contact the researchers via e-mail if you wish to view the results of the study.

If you have any questions about this study or require further information, please contact us using the contact information provided below. This study has been reviewed and received ethics clearance through the Research Ethics Board at Brock University (file # 08-236). If you have any comments or concerns resulting from your participation in this study, please contact the Research Ethics Office at (905) 688-5550 Ext. 3035, reb@brocku.ca. **Thank you in advance for your assistance with our research and your interest in this project!**

Yours sincerely,

Dr. Dawn Good	Julie St.Cyr-Baker	Angela Dzyundzyak
Dawn.Good@brocku.ca	js01cb@brocku.ca	ad03cr@brocku.ca

Contact at (905) 688-5550 ext. 3556

INDIVIDUAL DIFFERENCES IN PERSONALITY AND HEALTH: CONSENT FORM

I have read the information presented in the information letter about a survey being conducted by Dr. Dawn Good, Julie St. Cyr-Baker and Angela Dzyundzyak investigating individual differences in personality and health in the Psychology Department at Brock University. I have had the opportunity to ask any questions related to this study, to receive satisfactory answers to my questions, and any additional details I wanted. I am aware that I may withdraw from the study at any time without consequences to myself by advising the student researcher of this decision.

With full knowledge, I agree, of my own free will, to participate in this study.

[ ] I have read and understand the above information regarding this study.

[ ] I have received a copy of this form.

[ ] I understand that I may ask questions at any time during the study and in the future.

[ ] I agree to participate in this study.

Participant's name (please print) _____

Participant's signature _____ **Date:** _____

[ ] I have explained this study to the participant

Researcher's signature _____ **Date:** _____

[ ] I acknowledge that I am participating in this study for _____ research participation hours in a psychology course (see below) and will not receive monetary payment for this study.

COURSE (please circle only one course):

PSYC 1F90 2P12 2P20 2F23 2P36 2P37 3P39 Other: _____

****PLEASE KEEP A COPY OF THIS CONSENT FORM FOR YOUR RECORDS****

This project has been reviewed and received ethics clearance through the Office of Research Ethics Board (REB File #: 08-236). If you have any pertinent questions regarding your rights as a participant, please contact the Research Ethics Officer via e-mail at reb@brocku.ca or you may call (905) 688-5550 extension 3035.

***** THANK YOU FOR YOUR PARTICIPATION!*****

Everyday Living Questionnaire (IDPH Study)

Please fill in or circle an answer for each of the following. If you have any questions regarding clarification please ask the researcher. Thank you for your time and effort!

1. How old are you? ____

2. Gender? M___ F____

3. What is the highest level of education you have presently completed?
   a Less than high school
   b. High School/Grade 12
   c. University 1 2 3 4 4+ (Years)
   d. College 1 2 3 4 4+

4. What is your major (e.g. English, Psychology, Science)? _____

5. Handedness
   a. Right
   b. Left
   c. Both

6. Have you ever been hospitalized for (circle any that apply):
   a. Fractures Y N
   b. Illness Y N
   c. Surgery Y N
   d. Neurological complications Y N
   e. Other Y N

   If you answered Y to any of the above, briefly please provide details:
   e.g. How old were you? How did it happen?
   _____
   _____
   _____

7. Have you ever been diagnosed with a neurological condition? Y N

8. Have you ever been diagnosed with a psychiatric condition? Y N

9. Are you currently taking any prescribed medications for a neurological or psychiatric condition? Y N

   a. If Yes, if you wish to disclose what medication please do so:_____

10. Have you ever sustained an injury to your head with a force sufficient to alter your consciousness (e.g. dizziness, vomiting, seeing stars, or loss of consciousness, or confusion)? Y N
    [If you answered **no** to this question you may move ahead to question 22]

If yes to question 10, please answer the following questions (if you have had more than one injury, please refer to the *most recent* time you injured your head):

11. If you answered yes to question 10, did you experience these symptoms for more than 20 minutes? Y N

12. Did you experience a loss of consciousness associated with the head injury? Y N

    i. If so, how long was the loss of consciousness?
        i. [ ] < 5 minutes
        ii. [ ] < 30 minutes
        iii. [ ] < 24 hours
        iv. [ ] < 1 week
        v. [ ] < 1 month
        vi. [ ] > 1 month

13. How did you injure your head?

    i. [ ] Motor vehicle collision
    ii. [ ] Sports-related injury
    iii. [ ] Falling
    iv. [ ] Other Please Specify:_____

14. Please briefly describe the incident during which the head injury occurred:

_____

_____

_____

15. Please answer the following questions:

    a. Did the head injury result in a concussion? Y N

    b. Did it require stitches? Y N

    c. Did you receive medical treatment for your injury? Y N

    d. Did you stay overnight at a medical care facility? Y N

e. Approximately how old were you at the time ___

f. How many months or year(s) have past since you hit your head? ___

16. Have you sustained *more than one* injury to your head with a force sufficient to alter your consciousness (e.g. dizziness, vomiting, seeing stars, or loss of consciousness, or confusion)? Y N

    a. **If yes**, how many times? ___

17. **If you answered yes to question 16**, did you experience these symptoms for more than 20 minutes? Y N

If you responded yes to question 16, please answer the following with respect to your *least recent* head injury:

18. Did you experience a loss of consciousness associated with the least recent head injury? Y N
    i. If so, how long was the loss of consciousness?
        i. [ ] < 5 minutes
        ii. [ ] < 30 minutes
        iii. [ ] < 24 hours
        iv. [ ] < 1 week
        v. [ ] < 1 month
        vi. [ ] > 1 month

19. How did you injure your head?

    i. [ ] Motor vehicle collision
    ii. [ ] Sports-related injury
    iii. [ ] Falling
    iv. [ ] Other Please Specify:_____

20. Please briefly describe the incident during which the least recent head injury occurred:

_____

_____

_____

21. Please answer the following questions:

    a. Did the head injury result in a concussion? Y N

b. Did it require stitches? Y N

c. Did you receive medical treatment for your injury? Y N

d. Did you stay overnight at a medical care facility? Y N

e. Approximately how old were you at the time ___

f. How many months or year(s) have past since you hit your head? ___

22. Have you ever experienced any other neural trauma (e.g. stroke, anoxia)? Y N
    a. **If yes**, please explain:

_____

_____

23. Do you smoke cigarettes? Y N

    If yes, approximately how many a day? _____

24. Do you regularly engage in consuming alcohol? Y N

    a. If yes, how many drinks per week do you consume? _____
    b. On average how many drinks would you consume in one outing? _____

25. Do you engage in recreational drug use (e.g. smoke marijuana, drop ecstasy, etc.)? Y N

26. Did you consume caffeine today (e.g. coffee, tea, energy drink, chocolate)? Y N

    a. **If yes**, how much?
       1 2 3 more than 3

    b. **If yes**, how much time has past since you last consumed caffeine today?

       Less than 1 hour More than 1 hour

27. Do you have sensitivity to perfumes or scents? Y N

    If yes, please rate your sensitivity:

    Not at all Very
    1 2 3 4 5 6 7 8 9

28. Do you have a valid driver's license? Y N

a. **If yes**, how long have you had a driver's license? 1-3 years 4-6 years 7+ years

29. Do you wear glasses or contacts? Y N

30. Do you live: on your own with roommates other

with parents/guardians with partner

31. How many university credits are you taking this semester?

0.5 1 1.5 2 2.5 3 3.5 4 4.5 5 5.5 6

32. On a scale of 1 to 9 rate your enjoyment of academics:

Not at all Very
1 2 3 4 5 6 7 8 9

33. Have you ever received any extra assistance during your educational history? Y N

Please circle any that apply and indicate when you received the assistance:
E = Elementary school H = High school U = University

a. Learning resource teacher E H U
b. Tutor E H U
c. Educational assistant E H U
d. Speech Language Pathologist E H U
e. Occupational Therapist E H U
f. Physical Therapist E H U
g. Other: Please Specify:_____ E H U

34. On a scale of 1 to 9 rate your enjoyment of your life situation:

Not at all Very
1 2 3 4 5 6 7 8 9

35. On a scale of 1 to 9 how stressful would you rate your day-to-day life:

Not at all Very
1 2 3 4 5 6 7 8 9

36. What extracurricular sport(s) did you play in:

    a. Elementary school:
        i. please describe (e.g. skating, baseball, etc.) – indicate if it was recreational (R) or competitive (C)

_____

        ii. How often did you play sports (per week)? _____

    b. High school:
        i. please describe/name the sport(s) – indicate if it was recreational (R) or competitive (C)

_____

        ii. How often did you play sports (per week)? _____

    c. Currently play sports in University
        i. please describe/name the sport(s) – indicate if it was recreational (R) or competitive (C)

_____

        i. How often do you play sports (per week)? _____

37. Do you exercise regularly? Y N

    a. **If yes**, how many times a week do you exercise?_____
    Please describe: _____

_____

38. When you ride a bike/skate/etc. do you wear a helmet? Y N not applicable

39. Do you regularly engage in relaxation techniques (e.g. deep breathing or yoga): Y N

    a. **If yes**, how many times a week do you engage in relaxation methods?_____
    Please describe: _____

40. Was last night's sleep typical for you? Y N

    If No, what was different (better, worse) ? _____

Why was it different? (stress, room temperature, noise, etc.)

_____

Please indicate how well you slept last night by circling a number:

Worst Possible 1 2 3 4 5 6 7 Best Possible

Sleep Sleep

Please indicate how you feel right now by circling a number:

Very Sleepy 1 2 3 4 5 6 7 Very Alert

41. Have you had anything out of the ordinary occur in the past day or so? Y N
If yes, please explain:

_____

_____

42. Circle any of the following that apply to your experience over the past 6 months:

Moved	Death of a family member
New Job	Death of a close friend
Loss of Job	Financial Difficulties
Loss of Relationship	Illness of someone close to you
New Relationship	Personal Illness/Injury
Reconciliation with partner	New Baby
Reconciliation with Family	Wedding/ Engagement (self)
Divorce (of self or parents)	Vacation
Entered 1st year at university	Disrupted Sleep

Question 42 format adapted from Holmes, T. & Rahe, R (1967). "Holmes-Rahe life changes scale". *Journal of Psychosomatic Research*, Vol. 11, 213-218.

43. Please indicate how your day has been so far by circling a number:

Calm	1	2	3	4	5	6	7	8	9	10	Busy
Pleasant	1	2	3	4	5	6	7	8	9	10	Unpleasant
NOT Stressful	1	2	3	4	5	6	7	8	9	10	VERY Stressful

PCSC Questionnaire

Please rate the frequency, intensity and duration of each of the following symptoms based on how they have affected you today according to the following scale:

FREQUENCY	INTENSITY	DURATION
1 = Not at all	1 = Not at all	1 = Not at all
2 = Seldom	2 = Vaguely present	2 = A few seconds
3 = Often	3 = Clearly present	3 = A few minutes
4 = Very often	4 = Interfering	4 = A few hours
5 = All the time	5 = Crippling	5 = Constant

	FREQUENCY	INTENSITY	DURATION
Headache	_____	_____	_____
Dizziness	_____	_____	_____
Irritability	_____	_____	_____
Memory Problems	_____	_____	_____
Difficulty Concentrating	_____	_____	_____
Fatigue	_____	_____	_____
Visual Disturbances	_____	_____	_____
Aggravated by Noise	_____	_____	_____
Judgment Problems	_____	_____	_____
Anxiety	_____	_____	_____

Gouvier, W. D., Cubic, B., Jones, G., Brantley, P., & Cutlip, Q. (1992). Postconcussion symptoms and daily stress in normal and head-injured college populations. *Archives of Clinical Neuropsychology, 7,* 193-211.

Emotional Intelligence Scale (Barchard, 2001)

On the following pages, there are phrases describing people's behaviors. Please use the rating scale below to describe how accurately each statement describes *you*. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age. So that you can describe yourself in an honest manner, your responses will be kept in absolute confidence. Please read each statement carefully, and then circle the number that corresponds to the number on the scale.

	1	2	3	4	5
	Very Inaccurate	Moderately Inaccurate	Neither Inaccurate nor Accurate	Moderately Accurate	Very Accurate

1	I express my affection physically.	1	2	3	4	5
2	I shout or scream when I'm angry.	1	2	3	4	5
3	I think about the causes of my emotions.	1	2	3	4	5
4	I listen to my feelings when making important decisions.	1	2	3	4	5
5	I like to watch children open presents.	1	2	3	4	5
6	I am deeply moved by others' misfortunes.	1	2	3	4	5
7	I am concerned about others.	1	2	3	4	5
8	I find it difficult showing people that I care about them.	1	2	3	4	5
9	I keep my feelings to myself, regardless of how unhappy I am.	1	2	3	4	5
10	I rarely think about how I feel.	1	2	3	4	5
11	I plan my life logically.	1	2	3	4	5
12	I dislike being around happy people when I'm feeling sad.	1	2	3	4	5
13	I am calm even in tense situations.	1	2	3	4	5
14	I feel little concern for others.	1	2	3	4	5
15	I laugh out loud if something is funny.	1	2	3	4	5
16	I can’t help but look upset when something bad happens.	1	2	3	4	5
17	I pay a lot of attention to my feelings.	1	2	3	4	5
18	I base my goals in life on inspiration, rather than logic.	1	2	3	4	5
19	I usually end up laughing if the people around me are laughing.	1	2	3	4	5
20	I am easily moved to tears.	1	2	3	4	5
21	I feel sympathy for those who are worse off than myself.	1	2	3	4	5
22	I have difficulty showing affection.	1	2	3	4	5
23	I find it difficult showing people that I'm angry with them.	1	2	3	4	5
24	I rarely analyze my emotions.	1	2	3	4	5
25	I believe important decisions should be based on logical reasoning.	1	2	3	4	5
26	I am unaffected by other people's happiness.	1	2	3	4	5
27	I am not easily disturbed by events.	1	2	3	4	5
28	I have no sympathy for criminals.	1	2	3	4	5
29	I express my happiness in a childlike manner.	1	2	3	4	5
30	I show my fear.	1	2	3	4	5
31	I am usually aware of the way that I'm feeling.	1	2	3	4	5
32	I plan my life based on how I feel.	1	2	3	4	5
33	I feel other people's joy.	1	2	3	4	5
34	I suffer from others' sorrows.	1	2	3	4	5
35	I sympathize with the homeless.	1	2	3	4	5

36	I keep my happy feelings to myself.	1	2	3	4	5
37	I keep my feelings to myself, regardless of how scared I am.	1	2	3	4	5
38	I am not in touch with my feelings.	1	2	3	4	5
39	I listen to my brain rather than my heart.	1	2	3	4	5
40	I rarely get caught up in the excitement.	1	2	3	4	5
41	I am unaffected by the suffering of others.	1	2	3	4	5
42	I look down on any weakness.	1	2	3	4	5
43	I sometimes laugh out loud when reading or watching TV.	1	2	3	4	5
44	I suspect that my facial expressions give me away when I feel sad.	1	2	3	4	5
45	I notice my emotions.	1	2	3	4	5
46	I believe emotions give direction to life.	1	2	3	4	5
47	I get caught up in the excitement when others are celebrating.	1	2	3	4	5
48	I am upset by the misfortunes of strangers.	1	2	3	4	5
49	I believe that criminals should receive help rather than punishment.	1	2	3	4	5
50	I rarely show my anger.	1	2	3	4	5
51	I often ignore my feelings.	1	2	3	4	5
52	I make decisions based on facts, not feelings.	1	2	3	4	5
53	I dislike children's birthday parties.	1	2	3	4	5
54	I rarely cry during sad movies.	1	2	3	4	5
55	I don’t like to get involved in other people's problems.	1	2	3	4	5
56	I hug my close friends.	1	2	3	4	5
57	I show my sadness.	1	2	3	4	5
58	I often stop to analyze how I'm feeling.	1	2	3	4	5
59	I listen to my heart rather than my brain.	1	2	3	4	5
60	I am strongly influenced by the good moods of others.	1	2	3	4	5
61	I would be upset if I saw an injured animal.	1	2	3	4	5
62	I believe that the poor deserve our sympathy.	1	2	3	4	5
63	I wish I could more easily show my negative feelings.	1	2	3	4	5
64	I rarely notice my emotional reactions.	1	2	3	4	5
65	I remain calm during emergencies.	1	2	3	4	5
66	I have little sympathy for the unemployed.	1	2	3	4	5
67	I show my feelings when I'm happy	1	2	3	4	5
68	I find it hard to stay in a bad mood if the people around me are happy.	1	2	3	4	5

Barchard, K. A. (2001). *Emotional and social intelligence: Examining its place in the nomological network.* Unpublished Doctoral Dissertation: Department of Psychology; University of British Columbia; Vancouver, BC; Canada.

SRP-III (Self Report of Psychopathy; Paulhaus et al., in press)

Please rate the degree to which you agree with the following statements about you.

1	2	3	4	5
Strongly Disagree	Disagree	Neutral	Agree	Strongly Agree

1. I'm a rebellious person.	1	2	3	4	5
2. I'm more tough-minded than other people.	1	2	3	4	5
3. I think I could "beat" a lie detector.	1	2	3	4	5
4. I have taken illegal drugs (e.g., marijuana, ecstasy).	1	2	3	4	5
5. I have never been involved in delinquent gang activity.	1	2	3	4	5
6. I have never stolen a truck, car or motorcycle.	1	2	3	4	5
7. Most people are wimps.	1	2	3	4	5
8. I purposely flatter people to get them on my side.	1	2	3	4	5
9. I've often done something dangerous just for the thrill of it.	1	2	3	4	5
10. I have tricked someone into giving me money.	1	2	3	4	5
11. It tortures me to see an injured animal.	1	2	3	4	5
12. I have assaulted a law enforcement official or social worker.	1	2	3	4	5
13. I have pretended to be someone else in order to get something.	1	2	3	4	5
14. I always plan out my weekly activities.	1	2	3	4	5
15. I like to see fist-fights.	1	2	3	4	5
16. I'm not tricky or sly.	1	2	3	4	5
17. I'd be good at a dangerous job because I make fast decisions.	1	2	3	4	5
18. I have never tried to force someone to have sex.	1	2	3	4	5
19. My friends would say that I am a warm person.	1	2	3	4	5
20. I would get a kick out of 'scamming' someone.	1	2	3	4	5
21. I have never attacked someone with the idea of injuring them.	1	2	3	4	5
22. I never miss appointments.	1	2	3	4	5
23. I avoid horror movies.	1	2	3	4	5
24. I trust other people to be honest.	1	2	3	4	5
25. I hate high speed driving.	1	2	3	4	5
26. I feel so sorry when I see a homeless person.	1	2	3	4	5
27. It's fun to see how far you can push people before they get upset.	1	2	3	4	5
28. I enjoy doing wild things.	1	2	3	4	5
29. I have broken into a building or vehicle in order to steal something or vandalize.	1	2	3	4	5
30. I don't bother to keep in touch with my family any more.	1	2	3	4	5
31. I find it difficult to manipulate people.	1	2	3	4	5
32. I rarely follow the rules.	1	2	3	4	5
33. I never cry at movies.	1	2	3	4	5

34. I have never been arrested.	1	2	3	4	5
35. You should take advantage of other people before they do it to you.	1	2	3	4	5
36. I don't enjoy gambling for real money.	1	2	3	4	5
37. People sometimes say that I'm cold-hearted.	1	2	3	4	5
38. People can usually tell if I am lying.	1	2	3	4	5
39. I like to have sex with people I barely know.	1	2	3	4	5
40. I love violent sports and movies.	1	2	3	4	5
41. Sometimes you have to pretend you like people to get something out of them.	1	2	3	4	5
42. I am an impulsive person.	1	2	3	4	5
43. I have taken hard drugs (e.g., heroin, cocaine).	1	2	3	4	5
44. I'm a soft-hearted person.	1	2	3	4	5
45. I can talk people into anything.	1	2	3	4	5
46. I never shoplifted from a store.	1	2	3	4	5
47. I don't enjoy taking risks.	1	2	3	4	5
48. People are too sensitive when I tell them the truth about themselves.	1	2	3	4	5
49. I was convicted of a serious crime.	1	2	3	4	5
50. Most people tell lies everyday.	1	2	3	4	5
51. I keep getting in trouble for the same things over and over.	1	2	3	4	5
52. Every now and then I carry a weapon (knife or gun) for protection.	1	2	3	4	5
53. People cry way too much at funerals.	1	2	3	4	5
54. You can get what you want by telling people what they want to hear.	1	2	3	4	5
55. I easily get bored.	1	2	3	4	5
56. I never feel guilty over hurting others.	1	2	3	4	5
57. I have threatened people into giving me money, clothes, or makeup.	1	2	3	4	5
58. A lot of people are "suckers" and can easily be fooled.	1	2	3	4	5
59. I admit that I often "mouth off" without thinking.	1	2	3	4	5
60. I sometimes dump friends that I don't need any more.	1	2	3	4	5
61. I would never step on others to get what I want.	1	2	3	4	5
62. I have close friends who served time in prison.	1	2	3	4	5
63. I purposely tried to hit someone with the vehicle I was driving.	1	2	3	4	5
64. I have violated my probation from prison.	1	2	3	4	5

Paulhaus, D.L., Hemphill, J.D., & Hare, R.D. (in press). *Self-Report Psychopathy scale Version III (SRP-III)*. Toronto: Multi-Health Systems.

BROCK UNIVERSITY

NEUROPSYCHOLOGY COGNITIVE RESEARCH LABORATORY

Debriefing Statement

Dear Participant:

Thank you for your participation in this research study. As you are aware, this research study is conducted by Dr. Dawn Good, Julie St. Cyr-Baker, and Angela Dzyundzyak in the Psychology Department at Brock University. This study is investigating individual differences in personality factors and health, especially in university students who have experienced a previous mild head injury (e.g. concussion).

Numerous young adults incur head injuries every year (CIHR, 2003) and of these the majority are mild head injuries (MHI; e.g. concussion). In addition, research has shown that between 25% to 45% of university students have sustained mild head injuries as a result of sports activities or accidental falls. Due to their vulnerable location most of these head injuries may involve the frontal lobes, albeit subtle and typically recoverable. Injury to the frontal lobes can result in a myriad of cognitive changes and altered emotional responses. We are investigating individual differences in university students who self-report MHI history as compared to those who do not. Previous work in our lab has shown that students with MHI, despite their highly competent status, may present with characteristics different from, albeit minimally and statistically, non-MHI cohorts. Further they report and exhibit lower levels of arousal and are relatively less stressed relative to their cohorts. The purpose of the current study is twofold a) provide current information regarding the prevalence and etiology of MHI in young adults and their reports of post-concussive symptoms, and; b) investigate possible individual differences in university students who have sustained a previous MHI. In addition, we aim to replicate the previous findings from our lab.

Your participation is important for us to be able to examine any individual differences in persons who have experienced a mild injury (or not), and have a greater understanding of the experiences of persons who have a prior history of having reported concussion. The findings from this study will contribute to research on epidemiology, incidence and prevalence of persons who have experienced head injury.
Please feel free to ask any questions regarding the study. You are invited to view the results of the study by its completion (February, 2010).

If you experienced any negative emotions (e.g. sensitive questions, cognitive demands) as a result of participating in this research study and wish to speak with a counsellor please contact: **Brock University Counselling Services, ST 400, (905) 688-5550 extension 3240** or the principal investigator Dr. Dawn Good, Registered Psychologist**.** If you feel you have not been treated according to the descriptions in this form, or your rights as a participant in research have been violated during the course of this project, you may contact the **Research Ethics Officer** at (905) 688-5550, extension 3035, please cite REB file #:08-236.

Thank you again for your time and participating in this study!!!

If you have any questions or concerns please feel free to contact us:

Dr. Dawn Good	Julie St. Cyr-Baker	Angela Dzyundzyak
(905) 688-5550 extension 3869	(905) 688-5550 extension 3556	(905) 688-5550 extension 3556
Dawn.Good@brocku.ca	js01cb@brocku.ca	ad03cr@brocku.ca

APPENDIX C

Subject: REB - 09-284 - Good - Accepted
From: Research Ethics Board <reb@brocku.ca>
Date: 09/07/2010 10:15 AM
To: "'dawn.good@brocku.ca'" <dawn.good@brocku.ca>, "'js01cb@brocku.ca'" <js01cb@badger.ac.brocku.ca>, "'sv05lz@brocku.ca'" <sv05lz@badger.ac.brocku.ca>
CC: "'Michelle McGinn'" <rebchair@brocku.ca>, Lori Ann Walker <lwalker@brocku.ca>

DATE: 7/9/2010

FROM: Michelle McGinn, Chair
Research Ethics Board (REB)

TO: Dawn Good, Psychology
Julie St. Cyr-Baker, Stefon van Noordt

FILE: 09-284 - Good
Faculty Research

TITLE: Emotion and Personality Differences

The Brock University Research Ethics Board has reviewed the above research proposal.

DECISION: Accepted

This project has received ethics clearance for the period of **July 9, 2010 to June 30, 2011** subject to full REB ratification at the Research Ethics Board's next scheduled meeting. The clearance period may be extended upon request. ***The study may now proceed.***

Please note that the Research Ethics Board (REB) requires that you adhere to the protocol as last reviewed and cleared by the REB. During the course of research no deviations from, or changes to, the protocol, recruitment, or consent form may be initiated without prior written clearance from the REB. The Board must provide clearance for any modifications before they can be implemented. If you wish to modify your research project, please refer to http://www.brocku.ca/research/policies-and-forms/forms-2 to complete the appropriate form Revision or Modification to an Ongoing Application.

Adverse or unexpected events must be reported to the REB as soon as possible with an indication of how these events affect, in the view of the Principal Investigator, the safety of the participants and the continuation of the protocol.

If research participants are in the care of a health facility, at a school, or other institution or community organization, it is the responsibility of the Principal Investigator to ensure that the ethical guidelines and clearance of those facilities or institutions are obtained and filed with the REB prior to the initiation of any research protocols.

The Tri-Council Policy Statement requires that ongoing research be monitored. A Final Report is required for all projects upon completion of the project. Researchers with projects lasting more than one year are required to submit a Continuing Review Report annually. The Office of Research Services will contact you when this form *Continuing Review/Final Report* is required.

Please quote your REB file number on all future correspondence.

MM/sp

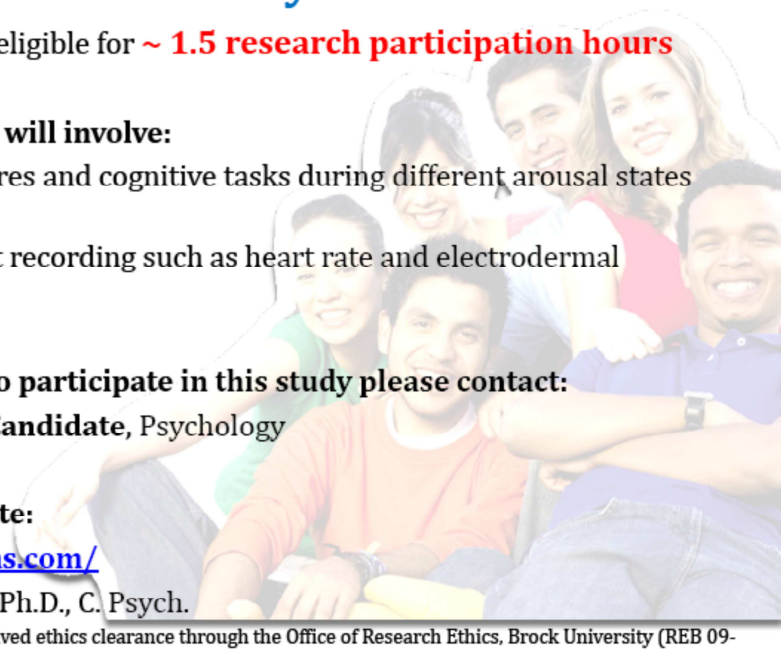
REB - 09-284 - Good - Accepted

Research Ethics Office
Brock University | Brock Research
Niagara Region | 500 Glenridge Ave. | St. Catharines, ON L2S 3A1
brocku.ca | T 905 688 5550 x3035 | F 905 688 0748

APPENDIX D

PARTICIPANTS NEEDED!
For research investigating
Emotion & Personality Differences

As a participant you will be eligible for **~ 1.5 research participation hours**



Participation in this study will involve:

•Completion of questionnaires and cognitive tasks during different arousal states (i.e. increased vigilance)

•Physiological measurement recording such as heart rate and electrodermal response

For more information or to participate in this study please contact:

Julie St. Cyr-Baker, Ph.D. Candidate, Psychology

js01cb@brocku.ca

Or sign up via SONA website:

http://brocku.sonasystems.com/

Supervisor: Dr. Dawn Good, Ph.D., C. Psych.

This study has been reviewed by and received ethics clearance through the Office of Research Ethics, Brock University (REB 09-284) 905-688-5550 ext. 3035

BROCK UNIVERSITY

Informed Consent Letter

Principal Student Investigator:
Julie St. Cyr-Baker, Ph.D. Candidate
Department of Psychology,
Brock University, St. Catharines, ON L2S 3A1
js01cb@brocku.ca
(905) 688-5550 ext. 3556

Principal Investigator:
Dr. Dawn Good, Ph.D., C. Psych.
Department of Psychology, Centre for Neuroscience
Brock University, St. Catharines, ON L2S 3A1
Dawn.Good@brocku.ca
(905) 688-5550, ext. 3869

Co-Investigator:
Stefon van Noordt, M.A. Candidate
Department of Psychology,
Brock University, St. Catharines, ON L2S 3A1
sv05lz@brocku.ca
(905) 688-5550 ext. 3556

You are being invited to participate in a research study. This study is investigating individual differences and personality factors that contribute to emotional experience. This research is being facilitated by Julie St. Cyr-Baker, Stefon van Noordt and Dr. Dawn Good. Your participation in this study is voluntary; there will be no negative consequences should you decline to participate at any time. If you choose to withdraw at any time during the **1.5 hour experimental session**, please inform the researcher and you will be credited with appropriate research participation hours reflecting your participation to that point. If you withdraw from the study before data collection is completed, your data will be omitted from the analysis and your response forms will be shredded. Please note that data cannot be removed after the session as responses are not linked to individuals. You also have the right to omit any answer(s) that you choose.

In this study you will be provided with two copies of this consent form, which will be read to you and, should you have any, feel free to ask questions about this research at that time. After reading the consent form, you will be asked to sign both copies, one for the researcher and one for your own records. If you decide to participate, you will next be asked to complete a demographic questionnaire and do various tasks. Each task will be described in detail as they are introduced. One of the tasks that you will be asked to participate in will involve viewing pictures that will include pleasant, unpleasant, and neutral scenes. In addition, physiological measures (i.e., heart rate, respiration, and electrodermal response) will be recorded via electrodes and other recording equipment. The application of the recording equipment will be described to you during the application process and will involve the placement of two electrodes on your fingers, placement of a pulse oximeter on your finger to record your heart rate, and respiration bands will be placed on your upper chest and lower abdomen. The areas of your skin involving contact with electrodes (i.e., your fingers) will be cleansed prior to, and after, electrode placement. Please advise the researcher if you have any dermal sensitivity. In order to reduce physical contact between yourself and the researcher you will be asked to assist in the placement and adjustment of the physiological recording apparatus. **You may ask questions at this time and at any time throughout the entire study.** Your

participation in this study will take approximately 1.5 hours. Note that you will be asked to complete various questionnaires. Some of the questions are personal and sensitive in nature. You will also be asked to provide background information about yourself such as sex, age, and level of education. Once you have completed the tasks, the specific purpose of the study will be explained and you will be provided a debriefing form.

Although there are no foreseeable risks for participating in this study it is possible that you may feel uncomfortable experiencing test performance anxiety and/or viewing pictures of an unpleasant nature may be uncomfortable. You are welcome to ask the researcher questions, or you may contact any of the counselling contact services (listed on your debriefing form), or contact the principal investigator, Dr. Dawn Good, Registered Psychologist, should you choose.

Your name will be associated only with this form. All information collected will be confidential and kept separately from this consent form, and coded by a number assignment. All consent forms, task data, and notes taken will be kept in a locked, secure lab at all times and will be destroyed after 5 years. Only Julie St.Cyr-Baker, Stefon van Noordt, Dr. Good, and research assistants will have access to this data. All research assistants have completed confidentiality agreements. In addition, any information gathered from this study used in discussions, publishable articles, or presentations will be summarized, preserving anonymity.

By participating in this study you may benefit from a better understanding of how psychological research is conducted. The information from this study will help with the completion of a Ph.D. thesis project and a Master's research project and will contribute to research on individual differences and personality factors that contribute to emotional experience. You will be invited to view the results of this study at its completion (by June 2011). Also, you may contact the researchers via e-mail if you wish to view the results of the study.

[ ] I have read and understand the above information regarding this study.
[ ] I have received a copy of this form.
[ ] I understand that I may ask questions in the future.
[ ] I agree to participate in this study.

Participant's name (please print) _____

Participant's signature _____ **Date:** _____

[ ] I have explained this study to the participant

Researcher's signature _____ **Date:** _____

[ ] I acknowledge that I am participating in this study for a maximum of 1.5 research participation hours in a psychology course (see below) and will not receive monetary payment for this study.

COURSE (please circle only one course):

PSYC 1F90 2P12 2P20 2F23 2P36 2P37 3P39 Other: _____

Participant's signature _____ **Date:** _____

****PLEASE KEEP A COPY OF THIS CONSENT FORM FOR YOUR RECORDS****

This project has been reviewed and received ethics clearance through the Office of Research Ethics Board (**REB File #: 09-284**). If you have any pertinent questions regarding your rights as a participant, please contact the Research Ethics Officer via e-mail at reb@brocku.ca or you may call (905) 688-5550 extension 3035. If you have questions at any time about the study or the procedures, or you experience adverse effects as a result of participating in this study, please feel free to contact us.

***** THANK YOU FOR YOUR PARTICIPATION!*****

Everyday Living Questionnaire (2009)

Please fill in or circle an answer for each of the following. If you have any questions regarding clarification please ask the researcher. Thank you for your time and effort!

1. How old are you? ____

2. Gender? M___ F____

3. What is the highest level of education you have presently completed?
   a Less than high school
   b. High School/Grade 12
   c. University 1 2 3 4 4+ (Years)
   e. College 1 2 3 4 4+

4. What is your major (e.g. English, Psychology, Science)? _____

5. Handedness
   a. Right
   b. Left
   c. Both

6. Have you ever been hospitalized for (circle any that apply):
   a. Fractures Y N
   b. Illness Y N
   c. Surgery Y N
   d. Neurological complications Y N
   e. Other Y N

   If you answered Y to any of the above, briefly please provide details:
   e.g. How old were you? How did it happen?
   _____
   _____
   _____

7. Have you ever been diagnosed with a neurological condition? Y N

8. Have you ever been diagnosed with a psychiatric condition? Y N

9. Are you currently taking any prescribed medications for a neurological or psychiatric condition? Y N

   a. If Yes, if you wish to disclose what medication please do so: _____

10. Have you ever sustained an injury to your head with a force sufficient to alter your consciousness (e.g. dizziness, vomiting, seeing stars, or loss of consciousness, or confusion)? Y N

[If you answered **no** to this question you may move ahead to question 22]

If yes to question 10, please answer the following questions (if you have had more than one injury, please refer to the *most recent* time you injured your head):

11. If you answered yes to question 10, did you experience these symptoms for more than 20 minutes? Y N

12. Did you experience a loss of consciousness associated with the head injury? Y N

    i. If so, how long was the loss of consciousness?
        i. [ ] < 5 minutes
        ii. [ ] < 30 minutes
        iii. [ ] < 24 hours
        iv. [ ] < 1 week
        v. [ ] < 1 month
        vi. [ ] > 1 month

13. How did you injure your head?

    i. [ ] Motor vehicle collision
    ii. [ ] Sports-related injury
    iii. [ ] Falling
    iv. [ ] Other Please Specify:_____

14. Please briefly describe the incident during which the head injury occurred:

_____

_____

_____

15. Please answer the following questions:

    a. Did the head injury result in a concussion? Y N

    b. Did it require stitches? Y N

    c. Did you receive medical treatment for your injury? Y N

    d. Did you stay overnight at a medical care facility? Y N

    e. Approximately how old were you at the time ___

    f. How many months or year(s) have past since you hit your head? ___

16. Have you sustained *more than one* injury to your head with a force sufficient to alter your consciousness (e.g. dizziness, vomiting, seeing stars, or loss of consciousness, or confusion)? Y N

    a. **If yes**, how many times? ___

17. **If you answered yes to question 16**, did you experience these symptoms for more than 20 minutes? Y N

If you responded yes to question 16, please answer the following with respect to your *least recent* head injury:

18. Did you experience a loss of consciousness associated with the least recent head injury? Y N
    i. If so, how long was the loss of consciousness?
        i. [ ] < 5 minutes
        ii. [ ] < 30 minutes
        iii. [ ] < 24 hours
        iv. [ ] < 1 week
        v. [ ] < 1 month
        vi. [ ] > 1 month

19. How did you injure your head?

    i. [ ] Motor vehicle collision
    ii. [ ] Sports-related injury
    iii. [ ] Falling
    iv. [ ] Other Please Specify:_____

20. Please briefly describe the incident during which the least recent head injury occurred:

_____

_____

_____

21. Please answer the following questions:

    a. Did the head injury result in a concussion? Y N

    b. Did it require stitches? Y N

    c. Did you receive medical treatment for your injury? Y N

    d. Did you stay overnight at a medical care facility? Y N

    e. Approximately how old were you at the time ___

f. How many months or year(s) have past since you hit your head? ___

22. Have you ever experienced any other neural trauma (e.g. stroke, anoxia)? Y N
    a. **If yes**, please explain:

_____

_____

23. Do you smoke cigarettes? Y N

    If yes, approximately how many a day? _____

24. Do you regularly engage in consuming alcohol? Y N

    a. If yes, how many drinks per week do you consume? _____
    b. On average how many drinks would you consume in one outing? _____

25. Do you engage in recreational drug use (e.g. smoke marijuana, drop ecstasy, etc.)? Y N

26. Did you consume caffeine today (e.g. coffee, tea, energy drink, chocolate)? Y N

    a. **If yes**, how much?
    1 2 3 more than 3

    b. **If yes**, how much time has past since you last consumed caffeine today?

    Less than 1 hour More than 1 hour

27. Do you have sensitivity to perfumes or scents? Y N

    If yes, please rate your sensitivity:

    Not at all Very
    1 2 3 4 5 6 7 8 9

28. Do you have a valid driver's license? Y N

    a. **If yes**, how long have you had a driver's license? 1-3 years 4-6 years 7+ years

29. Do you wear glasses or contacts? Y N

30. Do you live: on your own with roommates other

    with parents/guardians with partner

31. How many university credits are you taking this semester?

0.5	1	1.5	2	2.5	3	3.5	4	4.5	5	5.5	6

32. On a scale of 1 to 9 rate your enjoyment of academics:

Not at all								Very
1	2	3	4	5	6	7	8	9

33. Have you ever received any extra assistance during your educational history? Y N

Please circle any that apply and indicate when you received the assistance:
E = Elementary school H = High school U = University

a.	Learning resource teacher		E	H	U
b.	Tutor		E	H	U
c.	Educational assistant	E	H	U	
d.	Speech Language Pathologist	E	H	U	
e.	Occupational Therapist		E	H	U
f.	Physical Therapist		E	H	U
g.	Other: Please Specify:_____		E	H	U

34. On a scale of 1 to 9 rate your enjoyment of your life situation:

Not at all								Very
1	2	3	4	5	6	7	8	9

35. On a scale of 1 to 9 how stressful would you rate your day-to-day life:

Not at all								Very
2	2	3	4	5	6	7	8	9

36. Do you exercise regularly? Y N

a. **If yes**, how many times a week do you exercise?_____
Please describe: _____

---

44. When you ride a bike/skate/etc. do you wear a helmet? Y N not applicable

45. Do you regularly engage in relaxation techniques (e.g. deep breathing or yoga): Y N

a. **If yes**, how many times a week do you engage in relaxation methods?_____
Please describe: _____

46. Was last night's sleep typical for you? Y N

If No, what was different (better, worse) ? _____

Why was it different? (stress, room temperature, noise, etc.)

_____

Please indicate how well you slept last night by circling a number:

Worst Possible 1 2 3 4 5 6 7 Best Possible
Sleep Sleep

Please indicate how you feel right now by circling a number:

Very Sleepy 1 2 3 4 5 6 7 Very Alert

47. Have you had anything out of the ordinary occur in the past day or so? Y N
If yes, please explain:

_____

_____

48. Please indicate how your day has been so far by circling a number:

Calm 1 2 3 4 5 6 7 8 9 10 Busy
Pleasant 1 2 3 4 5 6 7 8 9 10 Unpleasant
NOT Stressful 1 2 3 4 5 6 7 8 9 10 VERY Stressful

49. Circle any of the following that apply to your experience over the past 6 months:

Moved	Death of a family member
New Job	Death of a close friend
Loss of Job	Financial Difficulties
Loss of Relationship	Illness of someone close to you
New Relationship	Personal Illness/Injury
Reconciliation with partner	New Baby
Reconciliation with Family	Wedding/ Engagement (self)
Divorce (of self or parents)	Vacation
Entered 1st year at university	Disrupted Sleep

Question 42 format adapted from Holmes, T. & Rahe, R (1967). "Holmes-Rahe life changes scale". *Journal of Psychosomatic Research*, Vol. 11, 213-218.

Emotional Intelligence Scale (Barchard, 2001)

On the following pages, there are phrases describing people's behaviors. Please use the rating scale below to describe how accurately each statement describes *you*. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age. So that you can describe yourself in an honest manner, your responses will be kept in absolute confidence. Please read each statement carefully, and then circle the number that corresponds to the number on the scale.

	1	2	3	4	5
	Very Inaccurate	Moderately Inaccurate	Neither Inaccurate nor Accurate	Moderately Accurate	Very Accurate

1	I express my affection physically.	1	2	3	4	5
2	I shout or scream when I'm angry.	1	2	3	4	5
3	I think about the causes of my emotions.	1	2	3	4	5
4	I listen to my feelings when making important decisions.	1	2	3	4	5
5	I like to watch children open presents.	1	2	3	4	5
6	I am deeply moved by others' misfortunes.	1	2	3	4	5
7	I am concerned about others.	1	2	3	4	5
8	I find it difficult showing people that I care about them.	1	2	3	4	5
9	I keep my feelings to myself, regardless of how unhappy I am.	1	2	3	4	5
10	I rarely think about how I feel.	1	2	3	4	5
11	I plan my life logically.	1	2	3	4	5
12	I dislike being around happy people when I'm feeling sad.	1	2	3	4	5
13	I am calm even in tense situations.	1	2	3	4	5
14	I feel little concern for others.	1	2	3	4	5
15	I laugh out loud if something is funny.	1	2	3	4	5
16	I can’t help but look upset when something bad happens.	1	2	3	4	5
17	I pay a lot of attention to my feelings.	1	2	3	4	5
18	I base my goals in life on inspiration, rather than logic.	1	2	3	4	5
19	I usually end up laughing if the people around me are laughing.	1	2	3	4	5
20	I am easily moved to tears.	1	2	3	4	5
21	I feel sympathy for those who are worse off than myself.	1	2	3	4	5
22	I have difficulty showing affection.	1	2	3	4	5
23	I find it difficult showing people that I'm angry with them.	1	2	3	4	5
24	I rarely analyze my emotions.	1	2	3	4	5
25	I believe important decisions should be based on logical reasoning.	1	2	3	4	5
26	I am unaffected by other people's happiness.	1	2	3	4	5
27	I am not easily disturbed by events.	1	2	3	4	5
28	I have no sympathy for criminals.	1	2	3	4	5
29	I express my happiness in a childlike manner.	1	2	3	4	5
30	I show my fear.	1	2	3	4	5
31	I am usually aware of the way that I'm feeling.	1	2	3	4	5
32	I plan my life based on how I feel.	1	2	3	4	5
33	I feel other people's joy.	1	2	3	4	5
34	I suffer from others' sorrows.	1	2	3	4	5
35	I sympathize with the homeless.	1	2	3	4	5

36	I keep my happy feelings to myself.	1	2	3	4	5
37	I keep my feelings to myself, regardless of how scared I am.	1	2	3	4	5
38	I am not in touch with my feelings.	1	2	3	4	5
39	I listen to my brain rather than my heart.	1	2	3	4	5
40	I rarely get caught up in the excitement.	1	2	3	4	5
41	I am unaffected by the suffering of others.	1	2	3	4	5
42	I look down on any weakness.	1	2	3	4	5
43	I sometimes laugh out loud when reading or watching TV.	1	2	3	4	5
44	I suspect that my facial expressions give me away when I feel sad.	1	2	3	4	5
45	I notice my emotions.	1	2	3	4	5
46	I believe emotions give direction to life.	1	2	3	4	5
47	I get caught up in the excitement when others are celebrating.	1	2	3	4	5
48	I am upset by the misfortunes of strangers.	1	2	3	4	5
49	I believe that criminals should receive help rather than punishment.	1	2	3	4	5
50	I rarely show my anger.	1	2	3	4	5
51	I often ignore my feelings.	1	2	3	4	5
52	I make decisions based on facts, not feelings.	1	2	3	4	5
53	I dislike children's birthday parties.	1	2	3	4	5
54	I rarely cry during sad movies.	1	2	3	4	5
55	I don’t like to get involved in other people's problems.	1	2	3	4	5
56	I hug my close friends.	1	2	3	4	5
57	I show my sadness.	1	2	3	4	5
58	I often stop to analyze how I'm feeling.	1	2	3	4	5
59	I listen to my heart rather than my brain.	1	2	3	4	5
60	I am strongly influenced by the good moods of others.	1	2	3	4	5
61	I would be upset if I saw an injured animal.	1	2	3	4	5
62	I believe that the poor deserve our sympathy.	1	2	3	4	5
63	I wish I could more easily show my negative feelings.	1	2	3	4	5
64	I rarely notice my emotional reactions.	1	2	3	4	5
65	I remain calm during emergencies.	1	2	3	4	5
66	I have little sympathy for the unemployed.	1	2	3	4	5
67	I show my feelings when I'm happy	1	2	3	4	5
68	I find it hard to stay in a bad mood if the people around me are happy.	1	2	3	4	5

Barchard, K. A. (2001). *Emotional and social intelligence: Examining its place in the nomological network.* Unpublished Doctoral Dissertation: Department of Psychology; University of British Columbia; Vancouver, BC; Canada.

BROCK UNIVERSITY
Emotion & Personality Differences Study
Debriefing Statement

Dear Participant:
Thank you for your participation in this research study. As you are aware, this research study was conducted by Julie St. Cyr-Baker, Stefon van Noordt, and Dr. Dawn Good in the Psychology Department at Brock University. This study is investigating individual differences and personality factors that contribute to emotional experience. The purpose of this study was to investigate whether increased emotional arousal can be maintained in university students who have/have not experienced a previous mild head injury. Also, individual differences in emotional interpretation (e.g., empathy, emotional processing) and responding will be examined.

This study examined whether induced emotional arousal interacts with a prior history of head injury and/or personality factors with respect to emotional experience. Previous research has shown that between 25% and 45% of undergraduate students have sustained a mild head injury and research from our lab (Brock University Neuropsychology Cognitive Research Lab) has shown that individuals with mild head injury are physiologically, and emotionally, underaroused (less stressed) relative to their peers. Our research has suggested that when higher levels of arousal are reported by individuals with mild head injury, their cognitive performance has shown to be optimally enhanced. Thus, we are examining whether certain stimuli can modify emotional arousal levels in persons who have/have not sustained a mild head injury. Ultimately, this will allow us to investigate whether or not these materials are particularly effective for possibly enhancing cognitive performance. We did not tell you about our interest in whether or not there is any indication of you having sustained a previous head injury (e.g., concussion) because there is published research that suggests that informing participants that head injury is a study variable of interest can influence subsequent performance (Suhr & Gunstad, 2002; 2005) such that individuals have preconceived impressions of what might be involved in having a head injury and, as a result, may behave in a way that either confirms these views (i.e., head injuries are often associated with limitations/changes in functional capacity and this may negatively affect how individuals approach and respond to task demands) or, at the least, in a way different from how they would have otherwise approached the task. In either case, the results would reflect how people react to having a head injury rather than simply performing the task. As a result, we did not advertise our interest in the variable, nor tell you about it prior to your participation.

We are also interested in how persons respond to and evaluate scenes that represent different events in life that may/may not be emotionally evocative and how personality factors (e.g., emotional profile) may influence these responses. To induce heightened arousal, you were asked to view and rate pictures of an unpleasant, pleasant, or neutral nature. This experimental manipulation for heightened arousal was used in order to provide, or otherwise produce, a heightened level of stress vigilance which is the precise effect being investigated.

Various psychological questionnaires were administered to examine emotional, personality and health factors. The State-Trait Anxiety Inventory (STAI, Spielberger, 1983) was administered to obtain an index of state and trait anxiety. The BarOn Emotional Quotient Inventory (EQ-i, Bar-on, 1997) was administered to provide an index of emotional-social capabilities. The Toronto Empathy Questionnaire (Spreng et al, 2009) was developed on the basis of several other empathy measures and was included to provide an index of empathy elicited on the basis of visual feedback. Mild head injury symptoms were assessed via the Post-Concussive Symptom Checklist (Gouvier et al., 1992) and demographic questionnaire. Also, heart rate, electrodermal activity, respiration, and blood pressure were recorded as physiological measures of emotional arousal response.

Your participation is important for us to be able to understand the relationships between subtle brain functions and everyday responses to social/environmental stimuli. Please feel free to ask any questions or contact us at anytime. You are invited to view the results of the study by its completion (June, 2011). Findings from this research study will form part of a Ph.D. thesis and a Master's research project and may be presented at conferences and/or in published format. Again, results will be anonymous as responses are not linked to individuals. It is important that you not discuss the procedures of participating in this study (until the end of term academic year 2010-2011) with other students as it may effect our results and we appreciate your cooperation.

If you experienced any negative emotions as a result of participating in this research study and wish to speak with a counsellor please contact: **Brock University Counselling Services, ST 400, (905) 688-5550 extension 3240** or the principal investigator Dr. Dawn Good, Registered Psychologist**. Should you like more information regarding head trauma please visit the following websites:** The Ontario Brain Injury Association (OBIA): http://www.obia.ca/ or The Ontario Neurotrauma Foundation (ONF): http://www.onf.org/. If you feel you have not been treated according to the descriptions in this form, or your rights as a participant in research have been violated during the course of this project, you may contact the **Research Ethics Officer** at (905) 688-5550, extension 3035, please cite REB file #:09-284.

Thank you again for your time and participating in this study!!!
If you have any questions or concerns please feel free to contact us:

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BROCK UNIVERSITY NEUROPSYCHOLOGY COGNITIVE RESEARCH LABORATORY

APPENDIX E

Emotion & Personality Study: IAPS Verbal Script 2011

Derived from:

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2005). *International affective picture system (IAPS): Instruction manual and affective ratings*. Technical Report A-6, The Center for Research in Psychophysiology, University of Florida.

Procedures

Viewing Phase – GET BASELINE RECORDING & SELF-REPORT OF AROUSAL BEFORE GIVING INSTRUCTIONS

To be said aloud to participant:

As I mentioned before, in this study we are interested in how people respond to different life events. In this next task you will be asked to view different pictures that represent a lot of different events that occur in life. For about the next 40 minutes you will be looking at different pictures projected on the screen in front of you, and you will be rating each picture in terms of how it made you feel while viewing it. There are no right or wrong answers, so simply respond as honestly as you can.

Now I'll describe the rating of the pictures in more detail. You will be using these scales to rate how you felt while viewing each picture (**give participant example ratings scale sheet**). You will make ALL ratings for EACH picture that you observe. The rating scales show four different kinds of feelings:

Arousal -Excited vs. Calm ranging from low to high arousal
Pleasant vs. Unpleasant
Intensity of emotional reaction to the image
Empathy ranging from no empathy to significant empathy

The first scale is the **arousal dimension** displayed here. At one extreme of the scale represents feeling stimulated, excited, frenzied, jittery, wide-awake, aroused. If you felt completely *aroused* while viewing the picture, use the mouse to indicate your response closer to the excited/high end of the scale. On the other hand, the other end of the scale represents feeling completely relaxed, calm, sluggish, dull, sleepy, unaroused. You can indicate you felt completely *calm* by choosing a '1' at the low end of the scale. You can represent intermediate levels by choosing any number along the scale **(demonstrate)**.

The **second scale is the pleasant-unpleasant scale**—at one end of the scale represents happy, pleased, satisfied, contented, or hopeful. So if you felt completely

happy while viewing the picture you can indicate this by indicating your response on the computer (i.e., press a 8 or 9). At the other end of the scale represents unhappy, annoyed, unsatisfied, melancholic, despaired, or bored. If you felt completely unpleasant while viewing the picture you can indicate your response on the computer by pressing the corresponding number. You can choose a number anywhere on the scale to represent your feeling of unpleasant or pleasant (demonstrate).

The **third scale** of feeling that you will rate is the **intensity scale**. At one end of the scale you have feelings characterized as strong and powerful. Please indicate feeling a strong intensity by choosing a number closer to the '*extremely intense*' end of the scale. The other end of this scale represents an intensity which is weak or absent (i.e., *not intense*). You can indicate that you felt the intensity was weak by indicating your response on the computer. Remember you can also represent your feelings between these endpoints **(demonstrate)**.

For the last scale you will be asked to provide a rating of **empathy – you will rate how much empathy the image elicits**. At one end of the scale you have feelings characterized as low or no empathy for those individuals in the photo. Please indicate feeling low or no empathy by choosing a number with the mouse. At the other extreme of this scale, you have feelings characterized as significant empathy for the person(s) in the photo. You can indicate feeling significant empathy by indicating your response on the computer. Remember you can also represent your empathy feelings between these endpoints **(demonstrate)**. If there are no persons in the photo please still provide a rating of how empathic the photo makes you feel.

Some of the pictures may prompt emotional experiences; others may seem relatively neutral. Your rating of each picture should reflect your immediate personal experience, and no more. Please rate each one *AS YOU ACTUALLY FELT WHILE YOU WATCHED THE PICTURE*.

The procedure will be as follows: It is important that your eyes be directed towards the screen when the pictures to be rated are shown –view the picture slide for the entire time it is on. You'll have only a few seconds to watch each picture. Please view the picture for the *entire* time it is on and make your ratings immediately *after* the picture is removed by indicating your response on the computer by pressing the appropriate key. After the picture is off, make your ratings on all dimensions as quickly as possible and get ready for the next picture. *After* each picture, you'll see each ratings scale and they will be shown on the screen **one at a time** and you can press the mouse on the computer to the number that corresponds to your rating of the picture for that scale. It is important that we have information for each of these pictures. There are no right or wrong answers; so *rate every picture on all dimensions*. It is very important *not* to dwell on your ratings of the pictures, since there will not be much time to give your response.

We are interested in your own *personal* ratings of the pictures.
Note that throughout this task recordings of your heart rate, electrodermal response and so forth will be taken so it is important that you remain relatively still and do not move the hand that is hooked up to the recording equipment. Thank you for your cooperation.

Are there any questions before we begin?

At the end of the 'viewing phase': Thank you for participating in this task. Now I'm going to take another recording from you so please try not to move more than necessary. [*Then* ask and **record self-report of arousal state on form**.]

(Note. Physiological Recording: Make sure that response markers are indicated throughout IAPS task)

Ratings Scales IAPS

Rate how arousing your emotional reaction is to this image

1	2	3	4	5	6	7	8	9

Low Moderate High

Rate how pleasant your emotional reaction is to this image

1	2	3	4	5	6	7	8	9

Unpleasant Moderate Pleasant

Rate how intense your emotional reaction is to this image

1	2	3	4	5	6	7	8	9

Not intense Moderate Extremely Intense

Rate how much empathy this image elicits

1	2	3	4	5	6	7	8	9

No Empathy Moderate Significant

APPENDIX F

Brock University
Research Ethics Office
Tel: 905-688-5550 ext. 3035
Email: reb@brocku.ca

Bioscience Research Ethics Board

Certificate of Ethics Clearance for Human Participant Research

DATE: 12/12/2012

PRINCIPAL INVESTIGATOR: GOOD, Dawn - Psychology

FILE: 12-084 - GOOD

TYPE: Masters Thesis/Project STUDENT: Julie Baker
SUPERVISOR: Dawn Good

TITLE: Emotion & Cognition Study

ETHICS CLEARANCE GRANTED

Type of Clearance: NEW Expiry Date: 12/31/2013

The Brock University Bioscience Research Ethics Board has reviewed the above named research proposal and considers the procedures, as described by the applicant, to conform to the University's ethical standards and the Tri-Council Policy Statement. Clearance granted from 12/12/2012 to 12/31/2013.

The Tri-Council Policy Statement requires that ongoing research be monitored by, at a minimum, an annual report. Should your project extend beyond the expiry date, you are required to submit a Renewal form before 12/31/2013. Continued clearance is contingent on timely submission of reports.

To comply with the Tri-Council Policy Statement, you must also submit a final report upon completion of your project. All report forms can be found on the Research Ethics web page at http://www.brocku.ca/research/policies-and-forms/research-forms.

In addition, throughout your research, you must report promptly to the REB:

a) Changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
b) All adverse and/or unanticipated experiences or events that may have real or potential unfavourable implications for participants;
c) New information that may adversely affect the safety of the participants or the conduct of the study;
d) Any changes in your source of funding or new funding to a previously unfunded project.

We wish you success with your research.

Approved:

Brian Roy, Chair
Bioscience Research Ethics Board

Note: Brock University is accountable for the research carried out in its own jurisdiction or under its auspices and may refuse certain research even though the REB has found it ethically acceptable.

If research participants are in the care of a health facility, at a school, or other institution or community organization, it is the responsibility of the Principal Investigator to ensure that the ethical guidelines and clearance of those facilities or institutions are obtained and filed with the REB prior to the initiation of research at that site.

APPENDIX G

Instructions for Home Sample Salivary Cortisol Collection

Collecting cortisol samples

Cortisol is a hormone that varies throughout the day. We can measure cortisol in saliva (spit) samples. We need saliva samples taken from you to compare with samples taken during your research participation session at Brock University.

The time the samples are collected is important. We have marked on the sample bag times for you to collect samples. Try to collect samples as close to the times specified as possible. There are three samples that you will provide:

SAMPLE 1

Evening prior to participation in research (Day 1) – please provide this sample at least 2 hours after eating and close to your bedtime (i.e., 10pm – 11:30pm); do not drink milk or caffeine within 1 hour of providing the sample.

Note. Lay out materials for morning samples so you remember to take them in the morning.

SAMPLE 2

Morning - Immediately upon awakening (Day 2) (take sample between 7:00-8:30am) – please do not brush your teeth, eat breakfast, or drink beverages until after sample 3 has been provided. It is best to keep the sample kit on your nightstand so you can provide the sample immediately upon waking. Note that approximately 45 minutes from this you will be providing another sample (i.e., sample 3).

SAMPLE 3

Morning - 45 minutes from when you collected sample 2 (Day 2)

Caffeine and milk products interfere with testing. We ask that you not have caffeine on the morning of sample collection until after all samples have been provided. Milk should be avoided for 30 minutes before sampling. Steroids can interfere with testing, too (e.g., prednisone).

Directions

1. Open bag and remove tube labelled either sample 1, sample 2, or sample 3.
2. Fill the tube approximately 2ml full by passively drooling into tube. If there are any bubbles in the saliva sample please fill above 2ml.
3. Close the tube tightly. Put the tube back into the bag and seal.
4. Write on the bag and the *record paper* the time the sample was collected.
5. Refrigerate samples until you bring them into the lab on the day of your testing session.

Note. If you have a difficult time producing saliva try thinking of your favourite food, biting into a lemon, or you may sip some plain water.

Everyday Living Questionnaire - Revised (Baker & Good, 2012)

Please fill in or circle an answer for each of the following. If you have any questions regarding clarification please ask the researcher. Thank you for your time and effort:

1. How old are you? _____
2. Gender? M____ F____
3. What is the highest level of education you have presently completed?
    a. Less than high school
    b. High School/Grade 12
    c. College 1 2 3 4 5+ (Years)
    d. University 1 2 3 4 5+ (Years)
4. What is the highest level of education your **father** has received?
    a. Less than high school
    b. High School/Grade 12
    c. College 1 2 3 4 5+ (Years)
    d. University 1 2 3 4 5+ (Years)
5. What is the highest level of education your **mother** has received?
    a. Less than high school
    b. High School/Grade 12
    c. College 1 2 3 4 5+ (Years)
    d. University 1 2 3 4 5+ (Years)
6. What is the overall average income your parents/guardians (If divorced, income of both parents combined)?
    a. Under $25,000
    b. $25,000 - $49,999
    c. $50,000 - $74,999
    d. $75,000 - $99,999
    e. $100,000 - $124,999
    f. $125,000 - $149,000
    g. $150,000 or more
7. What ethnicity do you identify most with:
    a. Hispanic
    b. Caucasian
    c. European
    d. African
    e. Chinese
    f. East Indian
    g. West Indian
    h. Japanese

i. Other

Specify: _____

8. In elementary school what were your career goals (what did you want to be when you grow up)?

_____
_____

9. In high school what were your career goals?

_____
_____

10. Have you switched your major during university? Yes No
If yes, please describe the change

_____

11. What is your major affiliated with (eg, Social Science, Humanities, etc.)
    a. Social Science
    b. Humanities
    c. Maths and Sciences
    d. Education
    e. Applied Health Science
    f. Business
    g. Undeclared
12. Are you currently pursuing an undergraduate or graduate degree?
    a. Undergraduate
    b. Graduate
    c. Not applicable
13. **If you answered 'a' to question 12**, are you planning on pursuing graduate studies after your undergraduate degree?
    a. Yes
    b. No
    c. Not sure yet
14. What do you currently hope to achieve with your education/what career do you want to pursue?

_____
_____
_____

15. Are you currently working while attending school? Yes No
16. **If you answered yes to question 15,** how many hours per week do you work?
    a. Less than 5
    b. 6 to 10
    c. 11 to 15

d. 16 to 20
e. More than 20 per week

17. **If you answered yes to question 15**, why do you work during school?
    a. Need the money
    b. Because you enjoy the job
    c. To fill spare time
    d. Other _____
18. Do you work a summer job when school is not in session? Yes No
19. **If you answered yes to question 18**, how many years have you had this job?
    _____
20. Which hand is your dominant hand (i.e. are you right or left-handed)?
    a. Right
    b. Left
    c. Both
21. Have you ever been hospitalized for (circle any that apply):
    a. Fractures Y N
    b. Illness Y N
    c. Surgery Y N
    d. Neurological Complications Y N
    e. Other Y N

    If you answered Y to any of the above, briefly please provide details (e.g. How old were you? How did it happen)

    _____
    _____
    _____
    _____
22. Have you ever been diagnosed with a neurological condition? Y N
23. Have you ever been diagnosed with a psychiatric condition? Y N
24. Are you currently taking any prescribed medications for a neurological or psychiatric condition? Y N
    a. If Yes, if you wish to disclose what medications please do so:
    _____
25. 6. [If female] Are you currently taking contraceptive pills or contraceptive injections (e.g., birth control pill or Depo-Provera)? Y N
26. Are you currently taking any performance enhancing drugs (e.g., anabolic steroids, Ritalin, etc.)?
    Y N

27. Have you ever sustained an injury to your head with a force sufficient to alter your consciousness (e.g. dizziness, vomiting, seeing stars, or loss of consciousness, or

confusion)? Y N

[If you answer **No** to this question you may move ahead to **question 41**]

If yes to question 27, please answer the following questions (if you have had more than one injury, please refer to the *most recent* time you injured your head):

28. If you answered yes to question 27, did you experience these symptoms for more than 20 minutes? Y N
29. Did you experience a loss of consciousness associated with the head injury? Y N
    i. If so, how long was the loss of consciousness?
        1. [ ] <5 minutes
        2. [ ] <30 minutes
        3. [ ] <24 hours
        4. [ ] <1 week
        5. [ ] <1 month
        6. [ ] >1 month
30. If applicable, where did you strike your head?
    a. Front of the head
    b. Right side of the head
    c. Left side of the head
    d. Other Provide brief details:

       _____
    e. I can't remember

31. How did you injure your head?
    i. [ ] Motor vehicle collision
    ii. [ ] Sports-related injury
    iii. [ ] Falling
    iv. [ ] Other Please Specify:

        _____
32. Please briefly describe the incident during which the head injury occurred:

    _____

    _____

    _____

    _____
33. Please answer the following questions:
    a. Did the head injury result in a concussion? Y N
    b. Did it require stitches? Y N
    c. Did you receive medical treatment for your injury? Y N
    d. Did you stay overnight at a medical care facility? Y N
    e. Approximately how old were you at the time? _____
    f. How many months or years have passed since you hit your head? _____

34. Have you sustained more than one injury to your head with a force sufficient to alter your consciousness (e.g. dizziness, vomiting, seeing stars, or loss of consciousness, or confusion)? Y N
35. **If you answered yes to question 34**, did you experience these symptoms for more than 20 minutes? Y N

If you responded yes to question 34, please answer the following with respect to your *least recent* head injury:

36. Did you experience a loss of consciousness associated with the head injury? Y N
    i. If so, how long was the loss of consciousness?
        1. [ ] <5 minutes
        2. [ ] <30 minutes
        3. [ ] <24 hours
        4. [ ] <1 week
        5. [ ] <1 month
        6. [ ] >1 month
37. If applicable, where did you strike your head?
    a. Front of the head
    b. Right side of the head
    c. Left side of the head
    d. Other Provide brief details:
       _____
    e. I can't remember

38. How did you injure your head?
    i. [ ] Motor vehicle collision
    ii. [ ] Sports-related injury
    iii. [ ] Falling
    iv. [ ] Other Please Specify:
        _____
39. Please briefly describe the incident during which the head injury occurred:
    _____
    _____
    _____
    _____
40. Please answer the following questions:
    a. Did the head injury result in a concussion? Y N

b. Did it require stitches? Y N
c. Did you receive medical treatment for your injury? Y N
d. Did you stay overnight at a medical care facility? Y N
e. Approximately how old were you at the time? _____
f. How many months or years have passed since you hit your head? _____

***********************If you were instructed to move ahead to question 41 please begin here*************

41. Have you ever experienced any other neural trauma (e.g. stroke, anoxia)? Y N
    a. **If yes,** please explain

       _____
       _____
       _____

42. Do you smoke cigarettes? Y N
    a. **If yes,** approximately how many a day? _____
43. Do you regularly engage in consuming alcohol? Y N
    a. If yes, how many drinks per week do you consume? _____
    b. On average how many drinks would you consume in one outing? _____
44. Do you engage in recreational drug use (e.g. smoke marijuana, drop ecstasy, etc.)? Y N
45. Did you consume caffeine today (e.g. coffee, tea, energy drink, chocolate)? Y N
    a. **If yes**, how much?
       1 2 3 more than 3
    b. **If yes**, how much time has passed since you last consumed caffeine today?
       Less than 1 hour More than 1 hour
46. Do you have sensitivity to perfume or scents? Y N
    If yes, please rate your sensitivity:
    Not at all Very
    1 2 3 4 5 6 7 8 9
47. Do you have a valid driver's license? Y N
    a. **If yes**, how long have you had a driver's license? 1-3 years 4-6 years 7+ years
48. Do you wear glasses or contacts? Y N
49. Do you live: on your own with roommates other
    with parents/guardians with partner

50. During elementary school, what were your average grades?
    a. A- to A+
    b. B- to B+
    c. C- to C+
    d. D- to D+

e. Other _____

51. During high school, what were your average grades?
    a. A- to A+
    b. B- to B+
    c. C- to C+
    d. D- to D+
    e. Other _____
52. Currently in University, what are your average grades?
    a. 90 to 100
    b. 80 to 89
    c. 70 to 79
    d. 60 to 69
    e. 50 to 59
    f. Other _____
53. How many university credits are you taking this semester?
    0 0.5 1 1.5 2 2.5 3 3.5 4 4.5 5 5.5 6
54. How many hours per week (on average) do you attend lectures/seminars/tutorials?
    Less than 5 6 – 9 10 – 12 13 – 16 17 – 20 21+
55. How many hours per week (on average) do you spend doing course readings for lecture/seminar/tutorial?
    Less than 3 4-6 7-9 10+
56. How many hours per week (on average) do you spend doing homework/assignments for lecture/seminar/tutorial?
    Less than 3 4-6 7-9 10+
57. On a scale of 1 to 9 rate your enjoyment of academics:
    Not at all Very
    1 2 3 4 5 6 7 8 9
58. Have you ever received any extra assistance during your educational history? Y N
    Please circle any that apply and indicate when you received the assistance:
    E = Elementary school H = High school U = University
    a. Learning resource teacher E H U
    b. Tutor E H U
    c. Educational assistant E H U
    d. Speech language pathologist E H U
    e. Occupational therapist E H U
    f. Physical therapist E H U
    g. Other: Please Specify _____ E H U
59. Have you ever been diagnosed or classified as having a Learning Disorder? Y N

60. Do you consider yourself a musician? Y N
61. Have you ever considered yourself to be a musician? Y N

62. If you answered yes to either question 60 or 61, did you play/perform:
    a. Professionally
    b. Recreationally
63. If you answered yes to either question 60 or 61, how long did you play/perform for?
    _____
64. What age did you start playing/performing at: _____ years
65. How often do you listen to music? _____ hours per week
66. Please indicate the type of music you listen to most often (can circle more than one):
    a. Country
    b. Classical
    c. Rock
    d. R&B
    e. Blues
    f. Independent
    g. Jazz
    h. Pop
    i. Electronic (house/dance)
    j. Folk
    k. Opera
    l. Other: Provide brief details:
       _____
67. On a scale of 1 to 9 rate your enjoyment of your life situation

Not at all								Very
1	2	3	4	5	6	7	8	9

68. On a scale of 1 to 9 how stressful would you rate your day-to-day life:

Not at all								Very
1	2	3	4	5	6	7	8	9

69. Do you consider yourself to be an athlete? Y N
70. What extracurricular sport(s) did you play in:
    a. Elementary school:
        i. Please describe (e.g. skating, baseball, etc.) – indicate if it was recreational (R) or competitive (C)

_____

        ii. How often did you play sports (per week)?
            _____
    b. High school:
        i. Please describe (e.g. skating, baseball, etc.) – indicate if it was recreational (R) or competitive (C)

_____

ii. How often did you play sports (per week)?

_____

c. University:

i. Please describe (e.g. skating, baseball, etc.) – indicate if it was recreational (R) or competitive (C)

_____

ii. How often did you play sports (per week)?

_____

71. In university, do you participate in any organized teams/sports? Y N

If no, please skip to **question 79**

If yes, please list the sports below and indicate if they are:

Community/Recreational	Intermural	Varsity
_____	_____	
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____		

72. How many consecutive years, including the current season, have you participated in each sport?

_____
_____
_____
_____

73. How many practices do you attend per week (per sport)?

_____
_____
_____
_____

74. How long in duration is the average practice (per sport)?

_____
_____
_____
_____

75. What does the typical practice consist of?

_____
_____
_____
_____
_____

_____
_____

76. In the last season, did you participate in any organized tournaments? Y N
If yes, for which sport(s)?
_____
If no, why not?
_____
77. Do you plan to attend any organized tournaments this season? Y N
If yes, for which sport(s)?
_____
If no, why not?
_____
78. Do any of your sports continue over the summer months when school is not in session? Y N
If yes, please describe any differences between the in season (school year) and off season (summer months) practices or workouts.
_____
_____
_____
_____
_____
_____
79. Do you exercise on a regular basis? Y N
80. Outside of organized practices for sports/teams, how many times per week do you exercise/work out?
_____
_____
81. Outside of organized practices for sports/teams, how long in duration is the average exercise/work out?
_____
_____
82. Outside of organized practices for sports/teams, what types of activities do you typically do to exercise/work out?
_____
_____
_____
_____
_____
83. Do you participate in any non-athletic extracurricular activities, clubs or groups?
_____

_____
_____
_____

If yes, how many hours a week (combined) do you spend at these activities?

_____

84. When you ride a bike/skate/etc. do you wear a helmet? Y N not applicable
85. Do you regularly engage in relaxation techniques (e.g. deep breathing or yoga): Y N
    a. **If yes**, how many times a week do you engage in relaxation methods? _____
    b. Please describe:

       _____

86. Was last night's sleep typical for you? Y N

    If No, what was different (better, worse)?

    _____

    Why was it different? (stress, room temperature, noise, etc.)

    _____
    _____

87. Please indicate how well you slept last night by circling a number:

    Worse Possible Sleep 1 2 3 4 5 6 7 Best Possible Sleep

88. Please indicate how you feel right now by circling a number

    Very Sleepy 1 2 3 4 5 6 7 Very Alert

89. Have you had anything out of the ordinary occur in the past day or so? Y N

    If yes, please explain:

    _____
    _____
    _____

90. Circle any of the following that apply to your experience over the past 6 months:

Moved	Entered 1st year at university
New Job	Death of a family member
Loss of Job	Death of a close friend
Loss of Relationship	Financial Difficulties
New Relationship	Illness of someone close to you
Reconciliation with Partner	Personal Illness/Injury
Reconciliation with Family	New Baby
Divorce (of self or parents)	Wedding/Engagement (self)

Vacation
Disrupted Sleep

91. Please indicate how your day has been so far by circling a number:

Calm	1	2	3	4	5	6	7	8	9 10 Busy
Pleasant	1	2	3	4	5	6	7	8	9 10 Unpleasant
NOT Stressful	1	2	3	4	5	6	7	8	9 10 VERY Stressful

Question 90 format adapted from Holmes & Rahe (1967)

Thank you for your time and consideration in completing this questionnaire! ☺

TEQ (Toronto Empathy Questionnaire – Spreng et al., 2009)

Below is a list of statements. Please read each statement *carefully* and rate how frequently you feel or act in the manner described. Circle your answer on the response form. There are no right or wrong answers or trick questions. Please answer each question as honestly as you can.

0	1	2	3	4
Never	Rarely	Sometimes	Often	Always

1	When someone else is feeling excited, I tend to get excited too	0	1	2	3	4
2	Other people's misfortunes do not disturb me a great deal	0	1	2	3	4
3	It upsets me to see someone being treated disrespectfully	0	1	2	3	4
4	I remain unaffected when someone close to me is happy	0	1	2	3	4
5	I enjoy making other people feel better	0	1	2	3	4
6	I have tender, concerned feelings for people less fortunate than me	0	1	2	3	4
7	When a friend starts to talk about his/her problems, I try to steer the conversation towards something else	0	1	2	3	4
8	I can tell when others are sad even when they do not say anything	0	1	2	3	4
9	I find that I am "in tune" with other people's moods	0	1	2	3	4
10	I do not feel sympathy for people who cause their own serious illnesses	0	1	2	3	4
11	I become irritated when someone cries	0	1	2	3	4
12	I am not really interested in how other people feel	0	1	2	3	4
13	I get a strong urge to help when I see someone who is upset	0	1	2	3	4
14	When I see someone being treated unfairly, I do not feel very much pity for them	0	1	2	3	4
15	I find it silly for people to cry out of happiness	0	1	2	3	4
16	When I see someone being taken advantage of, I feel kind of protective towards him/her	0	1	2	3	4

Spreng, R. N., McKinnon, M. C., Mar, R. A., & Levine, B. (2009). The Toronto Empathy Questionnaire: Scale development and initial validation of a factor-analytic solution to multiple empathy measures. *Journal of Personality Assessment*, *91*(1), 62–71. doi:10.1080/00223890802484381

Emotional Intelligence Scale (Barchard, 2001)

On the following pages, there are phrases describing people's behaviors. Please use the rating scale below to describe how accurately each statement describes *you*. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age. So that you can describe yourself in an honest manner, your responses will be kept in absolute confidence. Please read each statement carefully, and then circle the number that corresponds to the number on the scale.

	1	2	3	4	5
	Very Inaccurate	Moderately Inaccurate	Neither Inaccurate nor Accurate	Moderately Accurate	Very Accurate

1	I express my affection physically.	1	2	3	4	5
2	I shout or scream when I'm angry.	1	2	3	4	5
3	I think about the causes of my emotions.	1	2	3	4	5
4	I listen to my feelings when making important decisions.	1	2	3	4	5
5	I like to watch children open presents.	1	2	3	4	5
6	I am deeply moved by others' misfortunes.	1	2	3	4	5
7	I am concerned about others.	1	2	3	4	5
8	I find it difficult showing people that I care about them.	1	2	3	4	5
9	I keep my feelings to myself, regardless of how unhappy I am.	1	2	3	4	5
10	I rarely think about how I feel.	1	2	3	4	5
11	I plan my life logically.	1	2	3	4	5
12	I dislike being around happy people when I'm feeling sad.	1	2	3	4	5
13	I am calm even in tense situations.	1	2	3	4	5
14	I feel little concern for others.	1	2	3	4	5
15	I laugh out loud if something is funny.	1	2	3	4	5
16	I can’t help but look upset when something bad happens.	1	2	3	4	5
17	I pay a lot of attention to my feelings.	1	2	3	4	5
18	I base my goals in life on inspiration, rather than logic.	1	2	3	4	5
19	I usually end up laughing if the people around me are laughing.	1	2	3	4	5
20	I am easily moved to tears.	1	2	3	4	5
21	I feel sympathy for those who are worse off than myself.	1	2	3	4	5
22	I have difficulty showing affection.	1	2	3	4	5
23	I find it difficult showing people that I'm angry with them.	1	2	3	4	5
24	I rarely analyze my emotions.	1	2	3	4	5
25	I believe important decisions should be based on logical reasoning.	1	2	3	4	5
26	I am unaffected by other people's happiness.	1	2	3	4	5
27	I am not easily disturbed by events.	1	2	3	4	5
28	I have no sympathy for criminals.	1	2	3	4	5
29	I express my happiness in a childlike manner.	1	2	3	4	5
30	I show my fear.	1	2	3	4	5
31	I am usually aware of the way that I'm feeling.	1	2	3	4	5
32	I plan my life based on how I feel.	1	2	3	4	5
33	I feel other people's joy.	1	2	3	4	5
34	I suffer from others' sorrows.	1	2	3	4	5
35	I sympathize with the homeless.	1	2	3	4	5

36	I keep my happy feelings to myself.	1	2	3	4	5
37	I keep my feelings to myself, regardless of how scared I am.	1	2	3	4	5
38	I am not in touch with my feelings.	1	2	3	4	5
39	I listen to my brain rather than my heart.	1	2	3	4	5
40	I rarely get caught up in the excitement.	1	2	3	4	5
41	I am unaffected by the suffering of others.	1	2	3	4	5
42	I look down on any weakness.	1	2	3	4	5
43	I sometimes laugh out loud when reading or watching TV.	1	2	3	4	5
44	I suspect that my facial expressions give me away when I feel sad.	1	2	3	4	5
45	I notice my emotions.	1	2	3	4	5
46	I believe emotions give direction to life.	1	2	3	4	5
47	I get caught up in the excitement when others are celebrating.	1	2	3	4	5
48	I am upset by the misfortunes of strangers.	1	2	3	4	5
49	I believe that criminals should receive help rather than punishment.	1	2	3	4	5
50	I rarely show my anger.	1	2	3	4	5
51	I often ignore my feelings.	1	2	3	4	5
52	I make decisions based on facts, not feelings.	1	2	3	4	5
53	I dislike children's birthday parties.	1	2	3	4	5
54	I rarely cry during sad movies.	1	2	3	4	5
55	I don’t like to get involved in other people's problems.	1	2	3	4	5
56	I hug my close friends.	1	2	3	4	5
57	I show my sadness.	1	2	3	4	5
58	I often stop to analyze how I'm feeling.	1	2	3	4	5
59	I listen to my heart rather than my brain.	1	2	3	4	5
60	I am strongly influenced by the good moods of others.	1	2	3	4	5
61	I would be upset if I saw an injured animal.	1	2	3	4	5
62	I believe that the poor deserve our sympathy.	1	2	3	4	5
63	I wish I could more easily show my negative feelings.	1	2	3	4	5
64	I rarely notice my emotional reactions.	1	2	3	4	5
65	I remain calm during emergencies.	1	2	3	4	5
66	I have little sympathy for the unemployed.	1	2	3	4	5
67	I show my feelings when I'm happy	1	2	3	4	5
68	I find it hard to stay in a bad mood if the people around me are happy.	1	2	3	4	5

Barchard, K. A. (2001). *Emotional and social intelligence: Examining its place in the nomological network.* Unpublished Doctoral Dissertation: Department of Psychology; University of British Columbia; Vancouver, BC; Canada.

Revised Life Orientation Test (LOT-R)

Instructions:
Please answer the following questions about yourself by indicating the extent of your agreement using the following scale:

[0] = strongly disagree
[1] = disagree
[2] = neutral
[3] = agree
[4] = strongly agree

Be as honest as you can throughout, and try not to let your responses to one question influence your response to other questions. There are no right or wrong answers.

_____ 1. In uncertain times, I usually expect the best.
_____ 2. It's easy for me to relax.
_____ 3. If something can go wrong for me, it will.
_____ 4. I'm always optimistic about my future.
_____ 5. I enjoy my friends a lot.
_____ 6. It's important for me to keep busy.
_____ 7. I hardly ever expect things to go my way.
_____ 8. I don't get upset too easily.
_____ 9. I rarely count on good things happening to me.
_____ 10. Overall, I expect more good things to happen to me than bad.

Scoring:
1. Reverse code items 3, 7, and 9 prior to scoring (0=4) (1=3) (2=2) (3=1) (4=0).
2. Sum items 1, 3, 4, 7, 9, and 10 to obtain an overall score.

Note Items 2, 5, 6, and 8 are filler items only. They are not scored as part of the revised scale.

The revised scale was constructed in order to eliminate two items from the original scale, which dealt more with coping style than with positive expectations for future outcomes. The correlation between the revised scale and the original scale is .95.

Reference:
Scheier, M.F., Carver C.S., and Bridges, M.W. (1994). Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): A re-evaluation of the Life Orientation Test. *Journal of Personality and Social Psychology*, **67**, 1063-1078.

MORNINGNESS-EVENINGNESS QUESTIONNAIRE (MEQ) Horne & Ostberg (1976)

Instructions:

- Please read each question very carefully before answering.
- Please answer each question as honestly as possible.
- Answer ALL questions.
- Each question should be answered independently of others. Do NOT go back and check your answers.

1. What time would you get up if you were entirely free to plan your day?

5:00 – 6:30 AM	5
6:30 – 7:45 AM	4
7:45 – 9:45 AM	3
9:45 – 11:00 AM	2
11:00 AM – 12 NOON	1
12 NOON – 5:00 AM	0

2. What time would you go to bed if you were entirely free to plan your evening?

8:00 – 9:00 PM	5
9:00 – 10:15 PM	4
10:15 PM – 12:30 AM	3
12:30 – 1:45 AM	2
1:45 – 3:00 AM	1
3:00 AM – 8:00 PM	0

3. If there is a specific time at which you have to get up in the morning, to what extent do you depend on being woken up by an alarm clock?

Not at all dependent	4
Slightly dependent	3
Fairly dependent	2
Very dependent	1

4. How easy do you find it to get up in the morning (when you are not woken up unexpectedly)?

Not at all easy	1
Not very easy	2
Fairly easy	3
Very easy	4

5. How alert do you feel during the first half hour after you wake up in the morning?

Not at all alert	1
Slightly alert	2
Fairly alert	3
Very alert	4

6. How hungry do you feel during the first half-hour after you wake up in the morning?

Not at all hungry	1
Slightly hungry	2
Fairly hungry	3
Very hungry	4

7. During the first half-hour after you wake up in the morning, how tired do you feel?

Very tired	1
Fairly tired	2
Fairly refreshed	3
Very refreshed	4

8. If you have no commitments the next day, what time would you go to bed compared to your usual bedtime?

Seldom or never later	4
Less than one hour later	3
1-2 hours later	2
More than two hours later	1

9. You have decided to engage in some physical exercise. A friend suggests that you do this for one hour twice a week and the best time for him is between 7:00 – 8:00 am. Bearing in mind nothing but your own internal "clock", how do you think you would perform?

Would be in good form	4
Would be in reasonable form	3
Would find it difficult	2
Would find it very difficult	1

10. At what time of day do you feel you become tired as a result of need for sleep?

8:00 – 9:00 PM	5
9:00 – 10:15 PM	4
10:15 PM – 12:45 AM	3
12:45 – 2:00 AM	2
2:00 – 3:00 AM	1

11. You want to be at your peak performance for a test that you know is going to be mentally exhausting and will last for two hours. You are entirely free to plan your day. Considering only your own internal "clock", which ONE of the four testing times would you choose?

8:00 AM – 10:00 AM	4
11:00 AM – 1:00 PM	3
3:00 PM – 5:00 PM	2
7:00 PM – 9:00 PM	1

12. If you got into bed at 11:00 PM, how tired would you be?

Not at all tired	1
A little tired	2
Fairly tired	3
Very tired	4

13. For some reason you have gone to bed several hours later than usual, but there is no need to get up at any particular time the next morning. Which ONE of the following are you most likely to do?

Will wake up at usual time, but will NOT fall back asleep	4
Will wake up at usual time and will doze thereafter	3
Will wake up at usual time but will fall asleep again	2
Will NOT wake up until later than usual	1

14. One night you have to remain awake between 4:00 – 6:00 AM in order to carry out a night watch. You have no commitments the next day. Which ONE of the alternatives will suite you best?

Would NOT go to bed until watch was over	1
Would take a nap before and sleep after	2
Would take a good sleep before and nap after	3
Would sleep only before watch	4

15. You have to do two hours of hard physical work. You are entirely free to plan your day and considering only your own internal "clock" which ONE of the following time would you choose?

8:00 AM – 10:00 AM	4
11:00 AM – 1:00 PM	3
3:00 PM – 5:00 PM	2
7:00 PM – 9:00 PM	1

16. You have decided to engage in hard physical exercise. A friend suggests that you do this for one hour twice a week and the best time for him is between 10:00 – 11:00 PM. Bearing in mind nothing else but your own internal "clock" how well do you think you would perform?

Would be in good form	1
Would be in reasonable form	2
Would find it difficult	3
Would find it very difficult	4

17. Suppose that you can choose your own work hours. Assume that you worked a FIVE hour day (including breaks) and that your job was interesting and paid by results). Which FIVE CONSECUTIVE HOURS would you select?

5 hours starting between 4:00 AM and 8:00 AM	5
5 hours starting between 8:00 AM and 9:00 AM	4
5 hours starting between 9:00 AM and 2:00 PM	3
5 hours starting between 2:00 PM and 5:00 PM	2
5 hours starting between 5:00 PM and 4:00 AM	1

18. At what time of the day do you think that you reach your "feeling best" peak?

5:00 – 8:00 AM	5
8:00 – 10:00 AM	4
10:00 AM – 5:00 PM	3
5:00 – 10:00 PM	2
10:00 PM – 5:00 AM	1

19. One hears about "morning" and "evening" types of people. Which ONE of these types do you consider yourself to be?

Definitely a "morning" type	6
Rather more a "morning" than an "evening" type	4
Rather more an "evening" than a "morning" type	2
Definitely an "evening" type	0

List of Neuropsychological Measures (Study 3)

- **BarOn Emotional Quotient Inventory (Baron, 1997)**
  - The BarOn EQ-i is a 133 item self-report standardized index of socioemotional intelligence**.** Generally, the BarOn EQ-i is used to assess abilities involving: a) emotional expression, b) emotional awareness and perception, c) emotional regulation/management, d) adapting to change in both intra-and interpersonal situations, and e) expression of positive moods and overall motivation. The BarOn EQ-i has five major subscales (i.e., "*Intrapersonal, Interpersonal, Stress Management, Adaptability, and General Mood"*) and 15 subscales ("*Intrapersonal*: Self-Regard, Emotional Self-Awareness, Assertiveness, Independence, and Self-Actualization; *Interpersonal*: Empathy, Social Responsibility, Interpersonal Relationship; *Stress Management:* Stress Tolerance, and Impulse Control; *Adaptability:* Reality Testing, Flexibility, and Problem Solving; *General Mood:* Optimism and Happiness"). The questionnaire statements (e.g., "I'm in touch with my emotions; I'm aware of the way I feel") are rated on a 5-point scale (1 *very seldom or not true of me* to 5 *very often true of me or true of me*).

- **Subtests from the WAIS-IV (Weschler, 2008)**
  - **Matrix Reasoning:** participants are to provide the next logical solution to an equation. This task provides an index of logical reasoning and abstract thought.
  - **Digit Symbol-Coding:** participants are asked to draw designs to a matched number. This task assesses attention to detail and sequencing.
  - **Letter-number sequencing:** participants are asked to say letters and numbers in varying orders. This task assesses working memory capacity, sequencing ability, and attentional skills.

- **Advanced Clinical Solutions Supplement to the WAIS-IV and WMS-IV (Weschler, 2009)**
  - **Social Cognition**—Three subtests (Social Perception, Faces, and Names) index components of social cognition, such as distinguishing different types of affect; recognizing affect from faces and prosody; identifying sarcasm; the ability to describe others' intentions, and memory for faces and names**.** Only the Affect Recognition subscale was used and includes facial expressions of anger, fear, sadness, surprise, happiness and neutral expressions.

- **Subtests from the DKEFS (Delis, Kaplan, & Kramer, 2001)**

  - **Trails:** Participants are asked to draw lines to connect letters and numbers. This task assesses visual attention, visual scanning, sequencing ability, and response time.

- **Subtests from the Wide Range Achievement Test (WRAT-4; Wilkinson & Roberston, 2006)**
  - **Word Reading:** provides an index of word knowledge and decoding through identifying letters and recognizing words.

- **Rey Complex Figure Test (Osterrieth, 1944)**
    - Participants are asked to copy and reproduce a complex geometric design both immediately and after a 30 minute delay. This task assesses visuospatial memory abilities, sequencing, and attention to detail.

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